DISSERTATION ON

PREVALENCE OF HIV INFECTION IN TUBERCULOSIS PATIENTS AND THE PREDICTORS OF HIV CO-INFECTION

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CERTIFICATE

This is to certify that this dissertation entitled **"PREVALENCE OF HIV INFECTION IN TUBERCULOSIS PATIENTS AND THE PREDICTORS OF HIV CO-INFECTION"** submitted by **Dr.BATHRAGIRI. M,** appearing for Part II M.D. Branch I General Medicine Degree examination in September 2006 is a bonafide record of work done by him under my direct audience and supervision in partial fulfillment of regulations of the Tamil Nadu Dr. M.G.R. Medical University, Chennai. I forward this to the Tamil Nadu Dr. M.G.R. Medical University, Chennai, Tamil Nadu, India.

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DECLARATION

I solemnly declare that the dissertation titled **" PREVALENCE OF HIV INFECTION IN TUBERCULOSIS PATIENTS AND THE PREDICTORS OF HIV CO-INFECTION "** is done by me at Madras Medical College and Government General Hospital, Chennai during 2005-2006 under the guidance and supervision of Prof.K.CHANDRA, M.D.

The dissertation is submitted to The Tamilnadu Dr.M.G.R. Medical University towards the partial fulfilment of requirements for the award of M.D. Degree (Branch I) in General Medicine.

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CONTENTS

INTRODUCTION

Tuberculosis continues to be a major public health problem in India. 30 percent of the population are infected with tubercle bacilli (4). The factors attributed for this are poor quality of life, poor housing, overcrowding, population explosion, undernutrition, lack of education, large families, early marriage, lack of awareness.

Added to these factors is the increasing incidence of HIV infection in developing countries. As HIV infection decreases cell mediated immunity, it predisposes to active tuberculosis in previously infected people. So HIV infection can increase the morbidity and mortality due to active tuberculosis and the socio economic burden.

Even in developed countries where tuberculosis was under control, with increasing incidence of HIV infection there is resurgence of tuberculosis.

Tuberculosis appears early in the course of HIV infection before the appearance of other manifestations of the disease and opportunistic infections. so the center for disease control, has advised to screen all tuberculosis patients for HIV confection.

In developing countries like India, where tuberculosis is endemic whether the same rule has to be followed remains unanswered. Studies

done in various parts of the country regarding seroprevalence of HIV infection shows prevalence ranging from 1% to 10%. The factors which predict the HIV co-infection in tuberculosis patients are also not clearly known.

Approximately one third of the 36 million HIV infected persons in the world are co-infected with M.Tuberculosis. 75% of these people reside in sub-Saharan Africa patients with HIV are at greater risk of reactivating latent infection (7-10% annual risk compared to 5-10% lifetime risk in an HIV - uninfected individual), of acquiring TB from an open contact (10-20% compared to 5-10%), of developing progressive primary disease (30-40% compared to 5-10%) and of developing disseminated, miliary on extrapulmonary disease (>60% compared to <25%)

The purpose of the study is to find out the seroprevalence of HIV infection in tuberculosis patients and to find out the predictors of HIV co-infection.

OBJECTIVES OF THE STUDY

1. To find out the seroprevalnece of HIV infection in Tuberculosis patients (Pulmonary and extrapulmonary) admitted in Govt. General Hospital, Chennai.

2. Ton find out the factors associated with HIV co-infection in Tuberculosis patients.

REVIEW OF LITERATURE

HISTORY OF TUBERCULOSIS

Tuberculosis is undoubtedly an ancient disease. Indeed, Man may have been affected by it ever since he evolved, as a species on this planet. Evidence of existence of tuberculosis has been found in the bones of prehistoric man, found in Germany & Egypt. (2500-1000 B.C.) (31). Description of what could be tuberculosis has also been noted in the ancient Hindu and Chinese writings. Hippocrates (460-377 B.C) also devoted some attention to tuberculosis. However he felt that attention to tuberculosis was a waste of time and such cases were a burden on the state.

The theory that tuberculosis is an infectious disease was conceived by Aristotle more than 2000 years before the discovery of tubercle bacillus. Villemin demonstrated in a series of classical experiments that tuberculosis is caused by a specific agent and that it can be transmitted from man to animals by inoculation of infected material (1865).Robert Koch applied himself to this task and in 1882 this elusive microbe was identified. Laennec in 1819 invented the stethoscope and described auscultation. Laennec and Bayle described the tubercles and added new knowledge, Rudolph Virchow, the founder of cellular pathology, described the development of caseation in

tuberculous tissue. In 1890, Koch produced tuberculin and described Koch's phenomenon. X-rays, discovered by Roentgen in 1895 were put into clinical use by 1904 and helped in diagnosis of pulmonary Tuberculosis. Calmette and Guerin produced alternated Bovine Bacillus after subculturing about two hundred and thirty time from 1908 till 1921.

HISTORY OF HUMAN IMMUNODEFICIENCY VIRUS

The Acquired Immune Deficiency Syndrome (AIDS) was first recognised in the United States in the summer of 1981, when the Center for Disease Control and Prevention (CDC) reported the unexplained occurrence of pneumocystis carinii pneumonia in five previously healthy homosexual men in Los Angeles and of Kaposi's sarcoma in 26 previously healthy homosexual men in New York and Los Angeles (3). Within months it was recognised in injection drug users (IDUs) and soon thereafter in recipients of blood transfusions and in haemophiliacs. Because of the disproportionate number of cases of AIDS among Haitians in the United States early in the epidemic, this group was incorrectly designated as a 'risk' group. As the epidemiological pattern of the disease unfolded, it became clear that a microbe transmissible by sexual contact and blood or blood products was the most likely etiologic agent of the epidemic.

In 1983, Human Immunodeficiency virus as isolated from a patient with lymphadenopathy, and by 1984 it was demonstrated clearly to be the causative agent of AIDS(3). The virus, which belongs to the lentivirus subfamily of the large family of retroviruses (Retroviridae) was formerly called lymphadenopathy associated virus (LAV), Human T lymphotropic virus (HTLV) type III and AIDS associated retrovirus (ARV). In 1985, a sensitive enzyme linked immunosorbent assay (ELISA) was developed (3). Seroprevalence studies when combined with monitoring of CD4 + T Cell counts as a parameter of immunosuppression, led to the realisation of the global pandemic of AIDS and that there is a broad spectrum of HIV disease ranging from asymptomatic infection and clinical latency to advanced clinical disease, the latter constituting AIDS. Today, it is clear that virtually every practicing physician in the country and worldwide will be required to have some degree of familiarity with the workup, diagnosis, management and specific treatment of HIV infect individuals.

Sequence Analysis has led to the estimate that HIV 1 was introduced into humans in the early 1930s.

Since 1981 AIDS has grown to be the second leading cause of death in Africa. (Accounting for over 20% of deaths). It is now recognised that the immune deficiency is a consequence of continuos

high level HIV replication leading to virus and immune mediated destruction of the key immune effector cell, the CD4 Lymphocyte.

HUMAN IMMUNODEFICIENCY VIRUS

The four recognised human retrovirus belong to two district groups. The human T lymphotropic viruses, HTLV I and HTLV II which are transforming retroviruses, and the Human Immunodeficiency Viruses, HIV 1 and HIV 2 which are cytopathic viruses. The most common cause of HIV disease throughout the world is HIV 1. HIV 2 was first identified in 1986 in West African patients and was originally confined to West Africa.

Each mature virion is spherical in shape it has a lipid membrane lined by a matrix protein and studded with glycoprotein gp 120 and gp 41 spikes surrounding a cone shaped protein core. This core houses two copies of the ssRNA genome and viral enzymes. The virus infects the CD4 cell in a complicated sequence of events involving initial attachment and receptor engagement through the viral gp 120 and the CD4 cell receptor. Other cell expressing the CD4 cell receptor and permissive to infection are monocytes and macrophages, follicular dendritic cells and microglial cells in the central nervous system. HIV can also infect astrocytes through CD4 cell receptor independent attachment and fusion involving the chemokine receptor CXCR4.

It has been calculated that each day more than 10^{10} virions are produced and 10^{9} CD4 cells destroyed. This represents a turnover of 30% of the total viral burden and 6-7% of the total body CD4 cells daily. A small percentage of T cells (<0.01%) enter a post - integration latent phase and represent the main reservoir of HIV within these cells there is ongoing low - level replication even when plasma levels of HIV are below the levels of detection as a result of antiretroviral treatment. They are important as sanctuary sites from anti viral therapy, continuing sources of virus.

On the basis of DNA sequencing HIV - 1 can be subdivided into group M (major world wide distribution) group O (coutlier, restricted to West Africa and divergent from group M) and group N (rare and highly divergent). Group M and O can be subdivided further into subtypes. There are 10 subtypes for group M lettered A-K. Subtype B predominates in North America. Australia and Europe, Subtype A in Africa, subtype C in South Africa and India and subtype E in Thailand.

HIV - 2 in an important, but separate retrovirus occurring in West Africa or countries with ties to the region and has at least five subtypes. The virus differs from HIV - 1, in the patients have lower viral loads, slower CD4 decline, Lower rates of vertical transmission and slower progression to AIDS (12 fold lower).

TRANSMISSION

1. Sexual : HIV infection is predominantly a sexually transmitted disease worldwide. Although more than 50% of case are due to homosexual transmission, heterosexual transmission is clearly the most common mode of infection world wide, particularly in developing countries (3).

The receiving partner is at a greater risk than the insertive partners, in both vaginal and anal intercourse.

There is approximately a 20-fold greater chance of transmission of HIV from a man to woman than from a woman to a man through vaginal intercourse (3), because female is the recipient partner in sexual intercourse, and due to various well known anatomical, biological and serological reasons.

2. Blood and Blood Products : HIV can be transmitted by blood and blood products, both among individual who share contaminated paraphernalia (needles and syringes) for injection drug use and in those who receive transfusion of blood and blood products. Hyperimmune gamma globulin, hepatitis B immunogolobulin, plasma derived hepatitis B vaccine and Rho immune globulin have not been associated with transmission of HIV infection (3). The procedures involved in processing the products either inactivate or remove the virus.

3. Occupational : The risk of HIV transmission following skin puncture from a needle or a sharp object that was contaminated with blood from a person with documented HIV infection is approximately 0.3 percent (3). The risk increases for exposures to blood from patients with advanced stage of disease, probably owing to the higher titer of HIV in the blood as well as to other factor such as the presence of more virulent strains of virus.

The risk associated with mucocutaneous exposure is 0.1%. Transmission of HIV through intact skin has not been documented. The risk of transmission from an infected health care worker to patients is extremely low.

4. Maternal-fetal transmission : HIV can be transmitted from mother to her fetus during pregnancy or delivery. This is an extremely important mode of transmission in developing countries where the proportion of infect women to men is approximately 1:1 (3). This occurs most commonly in the perinatal period. Factors associated with higher risk are advanced disease, low CD4 + T cell count, high level of viremia, Vit A deficiency, chorioamnitis and funisitis and acute primary HIV infection during pregnancy. Postnatal transmission through colostrum and breast milk has also been clearly documented. Risk attributable to breast feeding 7 to 22 percent.

Although virus can be identified from virtually every body fluid, there is no evidence that HIV transmission can occur as a result of exposure to tears, sweat and urine (3).

S.No.	Modes of Transmission	Efficacy	Source of infection
1.	Sexual intercoruse	0.1 - 1.0%	80 - 86%
2.	Blood transfusion	90 - 95%	3 - 5 %
3.	Perinatal	20 - 40 %	2 - 3 %
4.	Injectable drug users	0.5 - 1.0%	3 - 5%
5.	Needle Stick exposure	< 0.1%	

TUBERCULOSIS : PROBLEM STATEMENT

World

Using the trends in case notification it is estimated that there were 8.8 million new cases of Tuberculosis in 2002 of which 3.9 million were smear positive. The global incidence rate of tuberculosis (percapita) is growing at approximately 1.1 percent per year and the number of cases at 2.4 percent per year. During 2002 about 1.56 million people died of this disease and global burden of disease in terms of DALYS lost was about 34.73 million. According to WHO estimates there were 16-20

million cases of Tuberculosis worldwide in 2001. The global incidence rate of for Tuberculosis being 119 per lakh population with low income countries carrying most of the burden with the incidence rate of 197 per lakh population. Middle income countries are having the rate of about 85 per lakh population, and high income countries about 9 per lakh population.

It is estimated that estimated that about one-third of current global population is infected asymptomatically with tuberculosis of whom 5-10 percent will develop clinical disease during their Life Time.

Eight out of Ten of all those struck by Tuberculosis are in economically productive age group of 15-49 years. It kills more adults than any other infectious disease. The South East Asia Region countries carry 38 percent of global burden of tuberculosis with 3 million new cases and nearly 0.6 million deaths occuring every year.

HIV PROBLEM STATEMENT

World

According to the recent estimates, by the end of 2004, 25 million people had already died of AIDS. Counting both those who have died and those currently living with virus in the past 20 years 65 million people have been infected. AIDS had orphaned more than 13 million

children worldwide. In south and south east Asia and the pacific about 6.1 million adults and 800,000 children were newly infected with the adult prevalence of 0.6%.

Global notes of HIV / AIDS epidemic December 2004.

People newly infected with HIV in 2004	Total Adults Children < 15 years	4.9 million 4.3 million 640000
Number of people living with HIV / AIDS	Total Adults Children < 15 years	39.4 million 37.2 million 2.2 million
AIDS deaths during 2004	Total Adults Children < 15 years	3.1 million 2.6 million 510000
Total number of AIDS orphans since the beginning of the epidemic		13.2 million

Trends in AIDS incidence show important difference between regions of the world. More than 95% of new cases remain in developing counties. In Africa, HIV infection in women now out-number those in

men. The number of AIDS deaths in industrialised countries has recently been falling due to combined antiretroviral therapies. In South - East Asia Region, the number of reported cases continue to increase and is likely to do so well into the early part of 21st Century. India, Thailand, Myanmar report the majority of cases with HIV/AIDS in the region. 91% are in the age group of 15-49 yrs and 4.6% are children. The male to female ration is 4 to 1. Heterosexual contact is the predominant mode of spread (85%) followed by injection drug use (7%) and mother to child transmission (5%).

Regional HIV /AIDS statistics and features, December 2003.

Region	**Adults and children living with HIV/AIDS**	**Adults and children Newly infected with HIV in 2003**	**Adults Prevalence Rate**
Sub-Saharan Africa	26.6.million	3.2 million	8.0%
North Africa and middle East	600,00	55000	0.3%
South and southeast Asia	6.4 million	855000	0.6%
East Asia and pacific	1.0 million	210000	0.1%
Latin America	1.6 million	150000	0.6%
Caribbean	470000	62500	2.5%
Eastern Europe and Central Asia	1.5 million	230000	0.7%
Western Europe	600000	35000	0.3%
North America	995000	45000	0.65

Australia and New Zealand	15000	850	0.1%
Total	40 million	5 million	1.1%

Source : Joint united Nations programme on HIV/AIDS (UN AIDS)

India

India's epidemic seems to be following the so called type 4 pattern, first described in Thailand. the epidemic shifts from the highest risk group (Commercial sex workers, drug users) to bridge population (clients of sex workers, STD patients and partners of drug users) and then to general pouplation.

HIV estimates for the year 2004 have been worked out to be 5.134 million HIV infections in the adults population (15-49 years age group in the country). These estimate have been pegged up for 20% as range (as in previous years) to take care of unaccounted number of high risk groups and other age groups to provide upper range as 5.1 million HIV infections.

The cumulative number of AIDS cases reported as on July 2005 is 1,11,608 out of which 89% of cases, are in the age group of 15 to 44 years, the economically productive age group. Out of the total AIDS cases reported 73% are men and 27% are women.

One of the characteristic features of Indian HIV scenario is its heterogeneously. While the whole country is afflicted with HIV infection, its prevalence various from state - to state. The most populous states of India are least affected for e.g. Uttarpradesh, Bihar Rajasthan, Madhya Pradesh, Orissa etc., while state like Maharastra, Tamilnadu, Karnataka Andrapradesh, Manipur and Nagaland are the most affected. In these stated HIV infection has even percolated into the general population leading to intense mother to child transmission of HIV.

The states will be categorized as high, moderate or low based one following definition.

- **High prevalent states:**

  States where HIV prevalence in antenatal women is 1% or more

- **Moderate prevalent states:**

  States where the HIV prevalence in antenatal women is less than 1% and prevalence is STD and other high risk groups is 5% or more

Low Prevalent States

States where the HIV prevalence in antenatal women is less than 1% and HIV prevalence among STD and other high risk group is less than 5%.

Group I (High prevalence states)

Includes states like Maharashtra, Karnataka, Andra Pradesh, Manipur, Mizoram, Nagaland and Dader and Nager Haveli where HIV infection has crossed 1% or more in antenatal mother. ANC prevalence in Tamilnadu has come down to 0.63 in 2004 from 1.13 in 2001.

Group II (Moderate prevalence states)

Included state like Gujarat, Kerala, Rajasthan and Goa where HIV infection has crossed 5% or more among high risk group but the infection is below 1% in Antenatal mothers.

Group III (Low prevalence states)

Includes remaining states where the HIV infection in any of the high risk group is still less than 5% and is less than 1% among antenatal mothers.

The high risk group of population includes patients attending STI clinics and intravenous drug uses while low risk of population including mothers attending antenatal clinics.

The epidemic in Tamilnadu has clearly shifted from high risk groups to general population. It reflects the percolation of the infection to general population.

HIV PREVALENCE IN RELATION TO TUBERCULOSIS

In 1988, A systematic nationwide sampling of HIV infection prevalence was undertaken by the Centre for Disease Control (CDC). The median seropositivity rate in 4301 persons with Tuberculosis was 3.4%. The rates varied widely (from 0% to 46%) among clinics. The highest rate was reported from New York City (46%) followed by New York (34%), Boston (27%), Miami (24), Baltimore (13%). A similar study in 1990 showed a seropositivity rate of 7.5%. In 1991 it was 9.5%. The overall seroprevalence rate in these areas was 13% in 1989, 18% in 1990, and 21% in 1991. Among Asians the HIV prevalence was less than 1% throughout the period.

Kenya : 50% in districts with an early increase in notification rates and 29% in the other study districts (18).

In the Hlabisa district Tuberculosis programme, South Africa, the prevalence of human immunodeficiency virus infection among adults with Tuberclosis increased from 36.0 in 1993 to 65.9 in 1997 (29).

The WHO has estimated that worldwide there are probably approximately 10 million persons who are infected with both M. tuberculosis and HIV, nearly 80% of them being in Africa. The prevalence of HIV among Tuberculosis patients in Sub-Saharan African countries ranged from 20% to 67%.

INDIA

An increase in HIV prevalance represents a serious threat to tuberculosis control in India current estimates suggest that nearly 4.56 million persons in India are infected with HIV and that approximately 1.64 million of these individuals are also infected with tuberculosis. An additional 1,40,000 TB cases can be expected annually among tuberculin skin-Test positive HIV infected individuals - as per Government of India (2004) Annual Report 2003-04 ministry of Health and family welfare, New Delhi.

Department of pediatrics, Jawaharlal Nehru Medical College, AMU, Aligarh, UP India conducted study in 2000 showed a seroprevalance rate of 2% among children below 12 years of age with pulmonary and extrapulmonary tuberculosis.

Department of Medicine All India Institute of Medical Sciences, Ansari Nagar, New Delhi India conducted study - the HIV seropositivity among adult TB patients from AIIMS between 2000-2002. The seropositive in adult TB patients was 9.4%.

Tuberculosis Research Centre IC MR Chennai India conducted study to estimate seroprevalence of human immuno deficiency virus(HIV) infection among tuberculosis patients in Tamilnadu. The study was under taken in four centres district tuberculosis centre (DTC) Vellore, Tuberculosis sanatorium Pennathur (Vellore) District TB centre (DTC) Kancheepuram and the Govt. Thiruvotteswarar Tuberculosis Hospital (GTTH), Chennai in the northern part of Tamilnadu during 1998. **Results:** The over all HIV seroprevlance among TB patients was 4.7 percent. The highest HIV seropositivity rate was found among patients aged 30-39 years (10.6%) Sputum smear positively was 88 percent among the HIV negative and 83 percent among HIV positive tuberculosis patients.

Rajan Babu TB Hospital and infectious diseases hospital GTB Nagar Delhi conducted study-clinical profile of tuberculosis in patients with HIV infection / AIDS. The most common symptom in these patients was cough and expectoration, followed by fever and weight loss. Acid fast Bacilli AFB smear positivity was found in 21.4% patients. On chest skiagram, infiltrative lesions were commonly seen in

61.9% patients. Extra pulmonary tubercular manifestations were seen in 45.6% HIV / TB cases.

Similar study conducted by the AIDS cell, Institute of Mcirobiology, Madras Medical College, Madras from January 1991 to May 1993, Showed a rise from 0.77% in 1991 to 3.4% in 1993 (17).

A similar study conducted at Institute of Thoracic Medicine, Chetpet, Chennai in 1997 showed an incidence of 4% of HIV positivity among Pulmonary Tubeculosis Patients.

A similar study conducted at Govt. Hospital for Thoracic Medicine, Tambaram, showed an incidence of 9.6% in 2000(33).

MATERIALS AND METHODS

STUDY DESIGN

Cross sectional study of one hundred tuberculosis patients.

STUDY POPULATION

Patients admitted with suspected Tuberculosis in General Medical wards Govt. General Hospital, Chennai.

INCLUSION CRITERIA

1. Patients who have radiological or bacteriological evidence of active Pulmonary Tuberculosis.
2. Meningitis patients with CSF analysis suggestive of Tuberculous meningitis.
3. Patients with exudative ascites with laparoscopic evidence of peritoneal Tuberculosis.
4. Histologically proved tuberculous lymphadenitis patients.

EXCLUSION CRITERIA

1. Old inactive pulmonary tuberculosis patients
2. Patients whose HIV status is already known.

STUDY DURATION

October 2005 to January 2006.

The study was initiated in October 2005 for the purpose of finding the prevalence of HIV infection among tuberculosis patients and the factors associated with HIV infection in tuberculosis patients. One hundred tuberculosis patients admitted in general medicine wards were studied.

Detailed history was taken and physical examination done for each patient as shown in proforma.

All patients underwent the following investigations.

1. **Complete Hemogram :** TC, DC, ESR, Hb%

2. **Chest X-ray PA View :** This was taken at Radiology department, Govt. General Hospital and reported by two doctors independently.

3. **Mantoux test :**

0.1 ml of purified protein derivative (PPD) of the strain RT 23 containing 1 tuberculin unit is injected intradermaly into the forearm of patients and a wheal is raised. Results are read after 48 hrs. An

induration with horizontal diameter of more than 10 mm is taken as positive for non HIV patients and induration of more than 5 mm is taken as positive for HIV positive patients.

SPUTUM AFB

Sputum is collected in sterile test tubes on three consecutive days and stained with Ziehl - Neelsen technique (Carbol fuchsin, Loeffler's Methylene blue) and the slide were screened by microbiologist for he presence of Acid fast Bacilli.

Apart from these investigations,

Pleural fluid analysis, ascitic fluid analysis, Cerebrospinal fluid analysis were done for cell count, Cytology, AFB, Protein if necessary to demonstrate tuberculous etiology.

SCREENING FOR HIV ANTIBODIES

Counselling

The center for Disease Control (CDC) has published recommendations for HIV testing and counselling. Testing for evidence of HIV infection should always be accompanied by pre and post test counselling. The counsellor should be knowledgeable about this process. Because results of tests for HIV infection have profound consequences

and raise many questions, persons should given written informed consent for the testing procedure and should understand the choices implied by the test results(1).

Counselling should include information about the test, HIV infection and AIDS as well as risk behaviours associated with the transmission of HIV. Discussion should also include the consequences of a positive or negative result for the person being tested (Medical care, Pregnancy, Employment, insurance) and other (family, lovers, friends) as well as the need for appropriate followup in the event of postive test results. Explanation of equivocal results that require additional tests may be necessary. Even for a person whose result is negative, counselling is recommended to allay a false sense of security and to promote future risk reduction behaviours. If the clinical suspicion of infection is high, based on risk behaviour or symptoms, follow up testing should be recommended. In the case of suspected primary infection, referral should be made to a research site or a knowledgeable HIV healthcare provider even if the antibody test is negative(1).

All the patients included in the study underwent pre and post test counselling.

SCREENING TESTS USED FOR HIV INFECTION

Enzyme linked Immunosorbent Assay

Most commonly used test because of their relatively simple methodology, inherent high sensitivity as well as specificity and suitability for testing large numbers of samples particularly in blood testing centers. The most popular ELISA involves an indirect method in which antibody in serum of the patient is allowed to react with HIV antigen attached to a well of a 96 well microtiter plate or to a macroscopic beed that subsequently is placed in a plate well. The test kits used in the study are LAB SYSTEMS and DETECT-HIV.

Rapid Test

These can be performed in less than 30 minutes. When performed correctly, rapid HIV assays are accurate. They are easy to perform and have utility in developing countries where facilities may not be optimal, stable electricity may be unavailable and formal education programme for laboratories are absent. They produce a well - circumscribed colored dot on the solid phase surface if the test is positive. The test kits used in the study are Tridot and Immunocomb (Bispot) where both HIV I and HIV II can be detected.

STRATEGIES OF HIV TESTING IN INDIA (30)

WHO / GOI have evolved strategies to detect HIV infection in different population groups and to fulfill different objectives. The various strategies, so designated, involve the use of categories of tests in various permutations and combinations.

1. ELISA /Simple /Rapid tests (E/R/S) used in strategy, I, II & III.

2. Supplemental test like Western Blot and line immunoassay are used in problem cases. e.g. in cases of indeterminate / discordant results of E/R/S.

UNAIDS and WHO Recommendations for HIV Testing Strategies According to Test Objective and Prevalence of Infection in the Sample Population

Objective of testing	Prevalence of infection	Testing Strategy
Transfusion / Transplant safety	All prevalence	I
Surveillance	> 10% ≤ 10%	I II
Diagnosis: Clinical signs/Symptoms of HIV infection	> 30% ≤ 30%	I II
Asymptmatic	> 10% ≤ 10%	II III

STRATEGY I

Serum is subjected once to E/R/S for HIV. If negative, the serum is to be considered free of HIV and if positive, the sample is taken as HIV infected for all practical purposes. This strategy is used for ensuring donation safety (blood/blood products/organ, tissue, sperms etc). The unit of blood tested reactive (positive) is discarded. Donar is not informed.

STRATEGY II

A serum sample is considered negative for HIV if the first ELISA report is so, but if reactive, it is subjected to a second ELISA which utilises a system different from the first one. It is reported reactive only if the second ELISA confirms the report of the first. This strategy is used for surveillance and for diagnosis only if some AIDS indicator disease is present.

STRATEGY III

It is similar to strategy II, with the added confirmation of a third reactive ELISA test being required for a sample to be reported HIV positive. The test to be utilized for the first ELISA is one with the highest sensitivity and for the second and third ELISAs tests with highest specificity are to be used.

All the patients included in the study were screened for HIV with ELISA. If it is reactive it is confirmed with another ELISA of different system and rapid test. It all are positive the patient is considered to be HIV positive.

LIMITATIONS OF THE STUDY

Tuberculosis patients who were sick enough to be admitted in hospital only were included in the study. Patients whose general condition is satisfactory and treated as outpatient were not included in the study.

ETHICAL ISSUES INVOLVED IN THE STUDY

Decisions about medical care, pregnancy, sexual and injection drug use behaviours and career planning can be affected by the results of an HIV antibody test. The risk that HIV test results may become known must be recognised by testing counsellors, clinicians, public health policy makers and above all persons who are considering being tested. Unfortunately persons with HIV confront fear and pain from knowing they are infected and serious social, financial and emotional problems resulting from individual and institutional prejudices about people with HIV and AIDS.

Eviction, job loss, inability to buy or maintain health or life insurance and abandonment by family and friends are examples of some of the unfortunate consequences of being labeled as infected with the HIV virus. These events are not rare and can occur whenever confidentiality is violated or test results are requested by and released to employees, insurance companies or other individuals and groups.

RESULTS

Out to hundred patients studied 62 are male and 38 are female.

The age distribution of the patients are as follows:

Age group	No. of Patients
11-20	07
21-30	27
31-40	27
41-50	19
51-60	16
61-70	4

Married - 73

Unmarried - 27

Ratio - 2.7:1

CXR IN HIV POSITIVE CASES

Bilateral Diffuse infiltrates - 4

Right sided pleural effusion - 1

Right upper zone cavity with

Bilateral midzone infiltrates - 1

SPUTUM AFB

Sputum AFB	**Positive**	**Negative**
HIV Negative	28	66
HIV Positive	1	5
Total	**30**	**70**

MANTOUX TEST

Mantoux	**Positive**	**Negative**
HIV Negative	43	51
HIV Positive	0	6
Total	**43**	**57**

DIAGNOSIS IN HIV POSITIVE CASES

Bilateral Pulmonary Tuberculosis	:	5
Right tuberculous pleural effusion	:	1

DIAGNOSIS IN ALL CASES

Pulmonary Tuberculosis	:	66
Pleural effusion	:	11
Pneumothorax	:	01
Abdominal Tuberculosis	:	03
Tuberculous Meningitis	:	01
Miliary tuberculosis	:	04
Spinal Tuberculosis	:	01
Pulmonary tuberculosis with Pleural Effusion	:	07
Pulmonary tuberculosis with Meningitis	:	02
Pulmonary tuberculosis with Lymphadenitis	:	02

Pleural effusion with Meningitis	:	01
Tuberculous Meningitis with Tuberculoma	:	01

MODE OF TRANSMISSION IN HIV POSITIVE CASES

Sexual	:	6
Blood transfusion	:	Nil
Hospital acquired	:	Nil

DISCUSSION

In this study 6 out of 100 Tuberculosis patients were HIV positive.

All the HIV positive patients comes under the age groups 25 to 40. Only one was above 35 and rest were between 25-35.

Five of them are males and one is female. All of them were married.

The study conducted by Department of Medicine, All India Institute of Medical Sciences Ansari Nagar, New Delhi, India to identify HIV seropositivity among adult tuberculosis patients in Delhi between 2000-2002. Of the 555 patients with various forms of Tuberculosis 52 were found to be seropositive **(9.4%)**. Another study conducted by Department of Experimental Medicine, Tamilnadu Dr.MGR Medical University, Madras where well documented 112 pulmonary Tuberculosis patients were studied for the prevalance of HIV seropositivity by using two antibody screening tests along with western blot test. Ninteen of the pulmonary Tuberculosis patients were HIV seropositive **(16.96%)**.

In our study 6 out of 100 Tuberculosis patients were HIV positive **(6%).**

Tuberculosis Research Centre (ICMR) Chennai India conducted study as seroprevalance of human Immunodeficiency virus infection among Tuberculosis patients in Tamilnadu. The study was undertaken in four centres. District Tuberculosis Centre (DTC) Vellore, Tuberculosis Sanatorium Pennathur (Vellore), District TB Centre (DTC), Kancheepuram and the Government Thiruvotteswarar Tuberculosis Hospital (GTTH), Chennai in the northern part of Tamilnadu during 1997-1998. A total of 2361 newly diagnosed TB patients were registered in this study. HIV serology after pre-Test counselling was done along with sputum examination for acid positive bacillus by smear for all patients. The overall HIV seroprevalence among TB patient was **4.7** percent. In our study it was 6%.

The highest HIV seropositivity rate was found among patients aged **30-39 years (10.6%)** in above ICMR study. In our study all the HIV positive patients comes under the age Groups between **25 to 35** except only one patient was above **35**.

In above ICMR study sputum smear positivity was 88 percent among the HIV negative and 83% percent among HIV positive patients and concluded that Acid - fast smear microscopy is adequate for the diagnosis of Pulmonary Tuberculosis. But in our study, Among total 94 HIV negative patients, 81 patients having pulmonary disease, 28 patients were sputum AFB positive. Among 6 HIV positive patients, 5

patients having pulmonary disease, and only one patient was sputum AFB positive. From our study HIV positive people with pulmonary tuberculosis may have a higher frequency of negative sputum smears in advanced state of HIV infection. So confirming the diagnosis may require sputum culture.

The tuberculin skin test often fails to work in people who are HIV positive because it relies on measuring the response of a person's immune system. If the immune system has been damaged by HIV, it may not respond even though the person is infected with Tuberculosis therefore have a higher frequency of false negative tuberculin skin tests results.

Department of TB diseases, **KG Medical University CSM Medical** University, Lucknow, India conducted study to determine the prevalance of human immunodeficiency virus infection among tuberculosis patients and to compare the clinico-radiological spectrum of tuberculosis tuberculosis among HIV seropositive and sero-negative patients was carried out. A total of 1105 Radiologically and or bacteriologically confirmed patients of tuberculosis were screened for HIV infection during the years 1995 to 1997 and from 2000-01.

Out of a total 1105 patients screened 31 (2.8%) were found to be HIV seropositive. Tuberculin positivity was less among HIV

seropositive patients as compared to HIV seronegative patients (22.6% vs 76.4%) $p<0.001$.

But in our study among 94 HIV seronegative patients 43 patients having Tuberculin skin test positive **(45%)**, none of HIV positive having tuberculin test positive **(0%)**.

Above CSM Medical University study, Among HIV seropositive patients mid and lower zone involvement, exudative lesions and mediastinal lymphadenopathy was more common as compared to seronegative patients, they concluded that the presentation of Tuberculosis was more often atypical among these patients. Another study Rajan Babu T.B. Hospital and infectious Diseases Hospital, GTB Nagar, Delhi - showed infiltrative lesions were commonly seen in 61.9% patients, Extra pulmonary tubercular manifestations were seen in 45.6% of HIV-TB.

In our study pulmonary tuberculosis was present in 5 out 6 positive patients **(83%).** Among these 5 patients, 4 patients having Bilateral diffuse in filtration **(80%)** and one patient having cavity lesion **(20%)**. Among 6 patients with HIV positive, one patient have Extrapulmonary Tuberculosis **(17%).**

Calcutta school of Tropical Medicine, Calcutta studied the different aspect of human immunodeficiency virus infection and

tuberculosis co-infection. Here an out patients department based survey conducted in Calcutta and found that all contracted HIV infection by hetero sexual route and multiple sex partners. In our study all 6 HIV seropositive patients having hetero sexual route was found to be the major risk factor for HIV. And all of them having multiple sex partners. And one of them also had genital ulcer disease a cofactor facilitating HIV transmission.

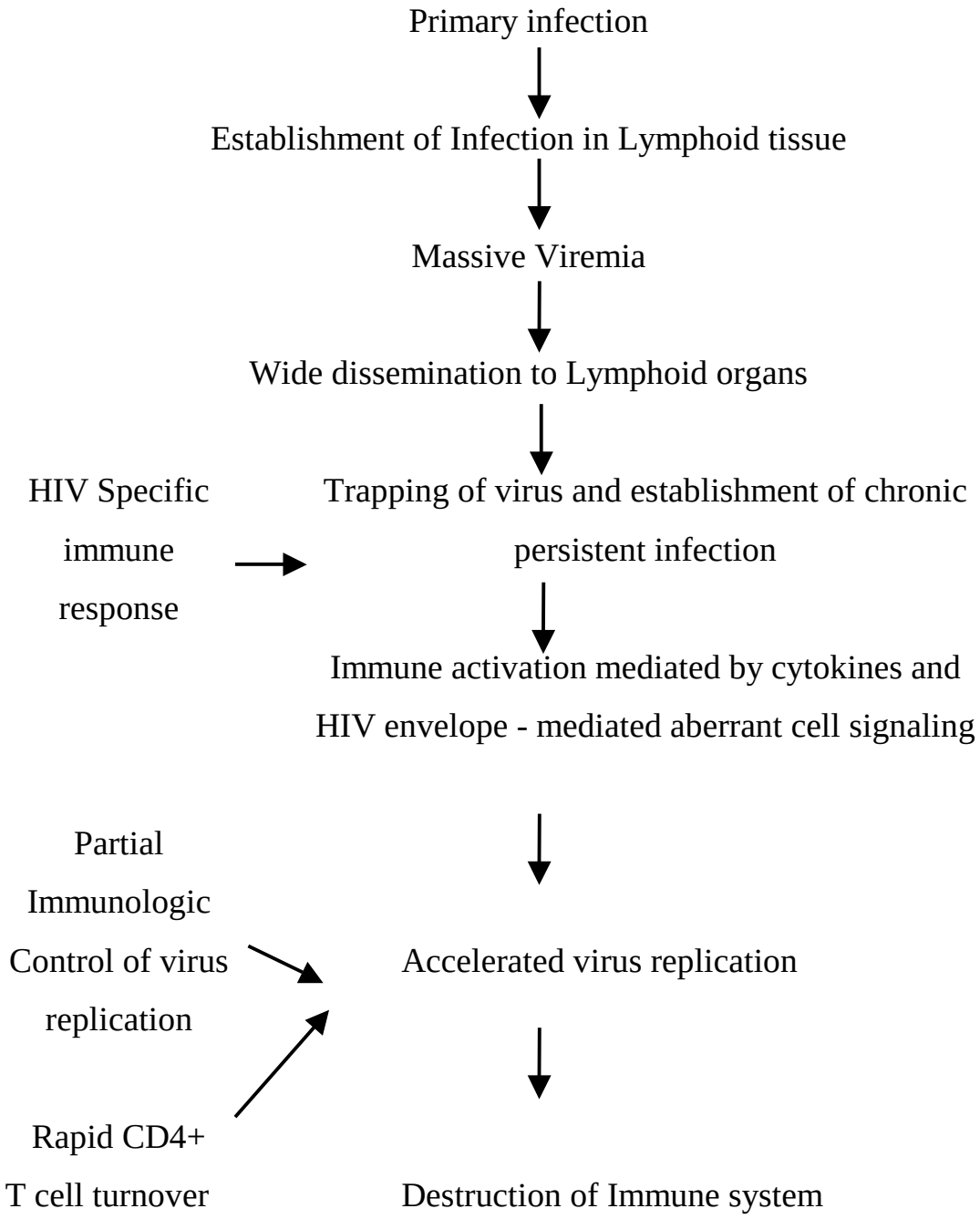
Rajan Babu TB Hospital and infections diseases Hospital GTB Nagar, Delhi conducted study of clinical profile of tuberculosis in patients with HIV infections / AIDS. Over a period of two years a total of 301 tuberculosis patients were suspected to HIV/AIDS coinfection and upon testing 42 patients were found to be HIV seropositive. Most of the study patients were manual labourers followed by trunk drivers. Sexual (hetero sexual) route was found to be the major risk factor for HIV / AIDS. The most common symptom in these patients was cough with expectration followed by fever and weight loss.

In out study all 6 HIV patients are Agriculture Labourers. All them having weight loss **(100%),** five out of 6 patients having cough with expectration **(83%)**. Four patient having fever **(66%)**. Three out of six patients having chronic diarrhea **(50%)**.

PATHOPHYSIOLOGY AND PATHOGENESIS OF HIV INFECTION

The hallmark of HIV disease is profound immunodeficiency resulting primarily from a progressive quantitative and qualitative deficiency of the subset of T lymphocytes refered to helper or inducer T cells. This subset has CD 4 molecules on their surface which serve as the primary cellular receptor for HIV. A coreceptor must be present together with CD4 for efficient fusion and entry of HIV - 1 into its target cells. They belong to the seven transmembrance - domain G protein - coupled family of receptors. Fusion or CXCR 4 is the coreceptor for T cell tropic strains of HIV - 1 and the ß - chemokine receptor CCR 5 is the coreceptor for macrophage - tropic strains of HIV - 1. The lymphy nodes are the major anatomic sites for the establishment and propagation of HIV infection.

PRIMARY INFECTION → DESTRUCTION OF IMMUNE SYSTEM



ESTABLISHMENT OF CHRONIC AND PERSISTENT INFECTION

HIV infection is relatively unique among human viral infections in that, despite the robust cellular and humoral immune responses that are mounted following primary infection, the virus is, with very few exceptions not cleared completely from the body. Rather a chronic infection developments that persists with varying degrees of virus replication for a median of approximately 10 years before the patient becomes clinically ill. It is this establishment of a chronic infection that is the hallmark of HIV disease. HIV has an extraordinary ability to mutate. HIV replication occurs throughout the course of HIV infection, even during clinical latency (3).

IMMUNO PATHOGENIC EVENTS DURING CLINICAL LATENCY

The level of CD4 + T cells in blood decrease gradually an progressively in HIV infected individuals. The slope of this decline together with level of the plasma viremia predicts well the pattern of the clinical course and the devlopment of advanced disease. Clinical latency does not mean disease latency, since progression is generally relentless during this period (3).

ADVANCED HIV DISEASE

After a variable period, usually measured in years, the CD 4 + T cell count falls below a critical level (<200 cells/µ lit) and the patient becomes highly susceptible to opportunistic disease. For this reason, the CDC case definition of AIDS was modified to include all HIV infected individuals with CD 4 + cells counts below this level. The depletion of CD 4 + cells continues to be progressive and unrelenting in this phase. It is not uncommon for CD 4 + T cell counts to drop as low as 10/µ L or even to zero. Patients who progress to this severest forms of immuno suppression usually succumb to opportunistic infections or neoplasms.

T CELL ABNORMALITIES

The defects are both quantitative and qualitative and involve virtually every limb of the immune system. Virtually all of the immune defects in advanced HIV disease can ultimately be explained by the quantitative depletion of CD 4 + T cells. One of the first abnormalities to be detected is a defect in response to remote recall antigens, such as tetanus toxoid and influenza, at a time when mononuclear cells can still respond normally to mitogenic stimulation.

The level of CD8 + T cells varies throughout the course of disease. Following the resolution of acute primary infection, CD 8 + T cells generally rebound to higher than normal levels, and they may

remain that way throughout the clinically latent stage of the disease. This CD 8 + T lymphocytosis may in part reflect the expansion of clones of HIV specific CD 8 + CT Lymphocytes. During the later stages of HIV infection there is a significant reduction in the numbers of CD 8 + T cells.

INTERACTION BETWEEN M.TUBERCULOSIS AND HIV INFECTION

The resurgence of Tuberculosis cannot be accounted for entirely by the epidemic of HIV infection, although the HIV epidemic clearly is a major factor. In addition to the biologic phenomenon of HIV infection, socioeconomic conditions such as homelessness, substance abuse and increase numbers of persons in institutions are playing an important role.

The importance of interaction between in Tuberculosis and HIV infection relates atleast 5 factors.

1. There is a high prevalence of tuberculosis in certain HIV groups.

2. Tuberculosis probably is the only HIV related infection that is transmitted from person to person whether or not the exposed person is HIV infected.

3. If diagnosed promptly and treated appropriately, Tuberculosis has a high likelihood of being cured.

4. Tuberculosis can be prevented in HIV infected populations and

5. Data from several source suggest that tuberculosis may accelerate the course of HIV disease.

Preexisting tuberculosis infection, indicating that endogenous reactivation was the dominant if not the only pathogenic mechanism for developing tuberculosis.

There are atleast four factors that influence the rate of tuberculosis among cohorts of persons with HIV infection (32).

1. Prevalence of latent infection with M. Tuberculosis in the population.

2. The likelihood of exposure of the cohort members to persons with infectious tuberculosis.

3. The severity of immune compromise among cohort members and thus, their likelihood of developing active tuberculosis if infected with the organism.

4. The use of isoniazid preventive therapy by cohort member will have an important effect in reducing the incidence of tuberculosis.

Variations among these factors probably account for the difference in reported rates of tuberculosis in different cohorts. Cohorts consisting largely of middle class homosexual/bisexual men or hemophiliacs would be expected to have low rates of Tuberculosis, whereas cohorts of injecting drug users living in inner cities would have higher rates. Thus, tuberculosis incidence data reported for one cohort may not apply to other groups, and there is no single figure that accurately defines the risk of tuberculosis in persons with HIV infection (32).

INFLUENCE OF HIV INFECTION ON THE PATHOGENESIS OF TUBERCULOSIS

Tuberculosis can develop either by direct progression from recently acquired infection or by reactivation of latent infection. Cell mediated immunity is the predominant mechanism by which a contained Tuberculosis is kept quiescent. Because HIV depresses cell mediated immunity, the likelihood of reactivation of latent Tuberculous infection leading to clinical Tuberculosis is greately increased.

MECHANISMS OF CD4 + LYMPHOCYTES DEPLETON & DYSFUNCTION (3)

HIV mediated direct cytopathicity (Single cell killing).

HIV mediated syncytia formation.

Virus specific immune responses.

HIV specific cytolytic T lymphocytes

Antibody - dependent cellular cytotoxicity (ADCC).

Natural killer cells.

Auto immune mechanisms

Anergy caused by inappropriate cell signaling via gp 120/CD4 interaction Superantigen mediated perturbation of T cell subsets.

Programmed cell death (apoptosis).

Defect in CD4 + T cell regeneration.

In the normal host, once the cell mediated immune response to infection with M. Tuberculosis develops, there is a low likelihood that new exogenous infection will be acquired. Because of the immune

defect inducted by HIV, a person may still be much more vulnerable to new infection. Thus, reinfection in persons with HIV infection may account for 'relapses' after successful completion of antituberculous therapy and also is a mechanism by which multi drug resistance can develop. It has been clearly demonstrated that once and HIV infected person becomes infected with M. tuberculosis, the infection can progress very rapidly to cause clinical disease. In a residential care facility for HIV infected persons in San Francisco, Tuberculosis occurred within 120 days in 11 of 31 (35%) residents exposed to a person with infectious tuberculosis.

Presumably because of the pathogenically of M. tuberculosis, tuberculosis tends to occur relatively early in the course of HIV infection. This is attested to by the findings of several groups that HIV seropositive patients with tuberculosis tend to have CD4 lymphocyte counts higher than patients with other 'opportunistic' infections like pneumocystic carinii pneumonia. In a study of 17 patients, Tuberculosis was the initial manifestation of HIV infection in all but two patients, and the median CD4 lymphocyte count was 354 / µl.

DIAGNOSIS OF TUBERCULOSIS INFECTION AND TUBERCULOSIS IN HIV INFECTED PATIENTS

Tuberculin skin testing

Among 94 HIV negative patients 43 patients showed positive mantoux test, 51 were negative. All 6 HIV positive patients were negative for tuberculin skin test. As would be expected, the tuberculin skin test commonly shows little or no reaction in persons with advanced HIV infection. However, in earlier stages of the infection, reactivity may be maintained. The ability to respond to tuberculin is an indicator of the status of cell mediated immunity that in turn is an indicator of the stage of HIV infection(32).

The prevalence of positive ($\geq$ 5 mm induration) tuberculin skin test decreased progressively as the CD4 cell count decreased.

In a population with a high prevalence of tuberculin reactors, a negative tuberculin skin test in an HIV infected person would likely be a false negative and that failure to react to the other antigens was a marker for severe immune compromise. Thus, such persons would be expected to have a high rate of tuberculosis. It would not be expected that this high risk of tuberculosis would apply to anergic persons from groups in which the prevalence of tuberculin reactivity is low(32).

In addition, the rate of reactivity to mumps and candida skin test antigens was related to the CD 4 count. It should be noted, however, that the prevalence of anergy was 42% among non-HIV infected injecting drug users and 12% among homosexual, bisexual men. These findings indicates the elusive nature of the definition and physiologic implications of 'anergy' and suggests that this finding should not weigh heavily in clinical decision-making.

Because of the frequency of blunted skin test responses,or anergy, it is recommended by American Thoracic Society and the CDC that a reaction of $\geq$ 5 mm induration to 5 tuberculin unit of purified protein derivative be regarded as indicative of tuberculosis is substantially increase in persons with reaction sizes $\geq$ 5 mm compared with the risk in persons with a 1 to 5 mm reactions (32)

Anergy testing has been recommended to determine if a negative tuberculin test is the result of immunosuppression or is truly negative, as well to provide information as to the stage of HIV disease. The antigens used include those made from candida organisms, mumps virus, and tetanus toxoid to which it is assumed that persons with intact cell mediated immunity will respond. But anergy may be found among persons who have no identifiable immunosuppression, thus limiting the clinical applicability of anergy testing.

CLINICAL FEATURES OF TUBERCULOSIS

Three HIV positive patients had history of chronic diarrhea.

In general examination 5 out of 6 were anaemic. Oral candidiasis and hairy leukoplakai were found in two patients. Dirty pigmented large tongue was present in one patient. One patient had extensive scabies. Hepatomegaly was present in two patient. Tinea versicolar was present in two patients and Icthyosis in one patient.

This vary considerably in HIV patients depending on the severity of the immunosuppression. Presumably because of the virulence of M. tuberculosis, tuberculosis may occur early in the course of HIV infection. In most series of majority of tuberculosis diagnoses have preceded the identification of another AIDS defining disease.

It has been shown that the earlier the tuberculosis develops, the more 'usual' is its clinical presentation, whereas the latter it occurs, the more atypical are its features. Tuberculosis in advanced HIV infection is frequently disseminated, has unusual radiographic manifestations and non reactive tuberculin skin tests. Lymphnode involvement, inducing intrathoracic adenopathy has been described frequently. A retrospective study reported a clear associations between low CD 4 cell counts and an increased frequency of extrapulmonary tuberculosis and intrathoracic adenopathy on chest radiograms. Conversely, pleural effusions were

more frequent in person with CD4 cell counts $\geq$ 200/µl. In this study one patient had pleural effusion.

Despite the increased frequency of unusual forms of tuberculosis in persons with HIV infection, standard, pulmonary disease tends to predominate in most series. One of the HIV positive patients had extra pulmonary tuberculosis in this study.

The clinical picture of TB in HIV + patient differs from in HIV - patient following ways: **(31)**

Clinical Picture	**HIV Negative**	**HIV Positive**
Constitutional Symptoms	+	+ Less specific
Extra Pulmonary disease	0-30%	60%
Pulmonary disease		
Subapical disease	Common	Uncommon
Cavittion	70%	0%
Lower lobe involvement	Rare	Common
Diffuse, Miliary Tuberculosis	Rare	Common
Hilar adenopathy	Rare	Common
Calcification	Common	Uncommon

RADIOGRAPHIC FINDINGS

CXR in HIV positive cases

Bilateral Diffuse infiltrates	-	4
Right sided pleural effusion	-	1
Right upper zone cavity with Bilateral midzone infiltrates	-	1

The atypical findings on chest radiographs of HIV infected patients who have Tuberculosis have received considerable emphasis. In retrospective studies, features that are not regarded as typical for pulmonary tuberculosis have been the norm. Lower lung zone or diffuse infiltrations commonly have been observed rather than the usual upper lobe involvement. Cavitation has been unusual and intrathoracic adenopthay, an unusual finding in immunocompetent adults with tuberculosis has been relatively frequent.

An evaluation of the radiographic course of treated Pulmonary tuberculosis in persons with HIV infection found that, in general, there was rapid improvement with little residual scarring after of therapy.

BACTERIOLOGIC AND HISTOLOGIC EXAMINATIONS

In this study 28 out of 94 HIV negative patients were positive for sputum AFB, one out of 6 HIV positive patients was positive.

Most reported series indicate that the prevalence of positive sputum smears and cultures in patients with pulmonary tuberculosis is approximately the same in HIV infected and noninfected persons. Potential high yield sources included lymphnodes, bonemarrow, urine and blood. In patients with more advanced HIV infection, mycobacterial infection does not produce classic granulomas. However, because, tuberculosis tends to occur when HIV diseases is less advanced, the ability to form granulomas may be intact. Thus, the finding of granulomas either in tissue sections or in cytologic preparations from needles aspiration biopsies should be interpreted as being more consistent with Tuberculosis (32).

RISK FACTOR ANALYSIS

All of 6 patients gave history of extra marital contact. The pattern of exposure varied in each patient. 6 patients had multiple contact with commercial sex workers. Non of the patients had homosexual contact. One patient had previous history of genital ulcer disease.

One person had surgery of haemarroid during which time, had 1 units of blood transfusion. No other risk factor could be indentified in that patients.

TREATMENT OF TUBERCULOSIS IN THE PRESENCE OF HIV INFECTION

Most reported series of patients with tuberculosis and HIV infection demonstrate a good response to anti tuberculosis treatment when regimens containing isoniazid and rifampin are used. Current recommendations state that for adult patients with HIV infection, treatment for tuberculosis should include isoniazid 300 mg/day, rifampin 600 mg per day. Pyrazinamide 20-30 mg/kg/day and ethambutol 15 mg/kg/day during the first 2 months of therapy. Isoniazid and rifampin should be continued for at least another 4 months, making the total duration of therapy at least 6 months. For patients judged to be potentially non complaint, therapy should be given under direct observation. This can be facilitated by twice weekly drug administration after and initial phase of daily treatment (32).

The rate of adverse reactions to antituberculosis drugs is greater in persons with HIV infection.They should be followed closely with appropriate laboratory and clinical monitoring. There has been no systematic evaluation of possible interactions of antibuberculosis drugs

with antiretroviral agents. Due to pharmacokinetic interactions, rifabutin should be substituted for rifampin in patient receiving the HIV protease in hibitors or non-nucleoside reverse transcriptase inhibitors. Both drugs should be avoided in patients receving Ritonavir. The antifungal agents Ketoconozole and fluconazole both have interactions with isoniazid and rifampin resulting in reduction in serum concentrations of the antifungal agents. In additional, ketoconazole interferes with absorption of rifampicin. Impaired absorption of antibuerculosis drug may also occur in patients with HIV infection, presumable as a consequence of gastrointestinal disease. For this reason if the response to treatment is suboptimal, serum drug concentrations should be measured. The incidence of multi drug resistant tuberculosis among HIV patients is increasing (32).

The ideal ART in the presence of Tuberculosis is

AZT + 3TC + EFAVIRANZ

OR

AZT + 3TC + SAQUINAVIR SGC WITH RITONAVIR BOOSTING

The Anti-Tuberculosis treatment could be varied depending on the CD4 cell count.

CD4 count	Drug regimen
More than 200	Complete Anti TB therapy first and then intiate ART therapy
Between 50 to 200	Intensive phase Initial two months - four Anti-TB drug therapy Continuation Phase Subsequent four months - Two Anti TB drug therapy. + ART together.
Less than 50	Both ART and above Anti-TB drug therapy can be started together

PREVENTION

The effectiveness of isoniazid preventive therapy in persons infected with both HIV and M. Tuberculosis has been substantiated. In addition to the benefit in preventing tuberculosis, it reduces the rate of progression to AIDS and also reduces the risk of death.

In view of these data, tuberculin testing should be performed as a routine part of management forpatietns with HIV infection. Patients with reactions of $\geq$5 mm should be recommended Isoniazid (300 mg per day) prophylaxis for 12 months (32).

SUMMARY

Among the hundred Tuberculosis patients studied 6 were positive for HIV infection. Duration of symptoms in HIV positive patients varied from 1 month 10 months. Three of them had chronic diarrhea.

Weight loss of more than 20 percent is a consistent finding in all the HIV positive patients though it is seen in most of tuberculosis patients to a lesser extent.

Among 6 HIV positive patients, 5 patients having pulmonary Tuberculosis. Extensive Pulmonary Tuberculosis was seen in 4 patients. Pleural effusion was seen in one patient. Cavitating lesions were seen in only one patient.

Hemogram and Erythrocyte sedimentation rat findings were similar in HIV positive and HIV negative patients. They were not contributory.

Among six human immune deficiency virus positive patients, five of them having pulmonary Tuberculosis. In these five patients one patient having sputum AFB positive **(20%)**. So confirming the diagnosis may require sputum culture.

Mantoux test was not positive (> 5mm) in any of the HIV positive patient. It was positive in only 43 of 94 HIV negative patients and it could not be depended upon for diagnosis of tuberculosis in both groups.

One of the HIV positive patients in the study had extra pulmonary tuberculosis.

CONCLUSION

The prevalence of HIV infection among Tuberculosis patients in this study is 6%.

As Tuberculosis is an early AIDS defining illness, it is mandatory to screen all tuberculosis patients for HIV infection and is being followed in western countries.

Though it is difficult to do the same in our country because of the high prevalence of Tuberculosis, prevalence of HIV infection among tuberculosis patients is rising. This study has showed a prevalence of 6%.

Pulmonary Tuberculosis continues to be the dominant form. Only one HIV Positive patients had extrapulmonary tuberculosis in this study.

The management of tuberculosis patients is not complete if they are not screened for HIV in the presence of following features.

1. Weight loss of more than 20%

2. History of multiple sexual partners, injection drug abuse, blood transfusion, Sexually Transmitted Diseases.

3. Atypical chest X-ray findings like lower lobe involvement, extensive parenchymal involvement, hilar mediastinal lymphadenopathy.

4. Extrapulmonary tubeculosis

5. Chronic diarrhea

BIBLIOGRAPHY

1. The AIDS knowledge Base, Third Edition, edited by P.T. Cohen, M.A. Sande, and P.A. Volberding, 1999.

2. AIDS, Biology, Diagnosis, Treatment and Prevention, Fourth Edition, Edited by Vincent T. Devita Jr. Semuel Hellman and Steren A Rosen berg, 1997.

3. Human Immunodeficiency Virus (HIV) Disease: AIDS and related disorders. Anthony S. Fanci H Clifford lane, in Harrison's Principles of Internal Medicine. 16th Edition, 2005, Volume 1, 1076-1139.

4. Park's textbook of preventive and social medicine, 18th edition, 2005, 146-160, 271-280.

5. Manual for control of Hospital Associated infections, National AIDS control Organisation, Ministry of Health and Family Welfare, Government of India.

6. International Journal of Tuberculosis and Lung Disease. Volume 3(8) 741-744.

7. Changing Trends in the management of HIV and Tuberculosis. K.C. Mohanty, Salil S. Bendre, Lung India. Vol. XVIII No.1, Jan-March 2000, 21-26.

8. A review of tuberculosis and the prospects for its elimination. Mechles Lauzards, David Aslikin Chest/117/5/May, 2000, 1455-1469.

9. Clinical and Radiographic predictors of the Etiology of Pulmonary Nodules in HIV infected patients. Robert M. Jasmer, Keith J. Edinburgh. Annemarie Thompson, Michael B. Gotway. Chest/117/4/April, 2000. 1023-1029.

10. Pattern of opportunistic pulmonary infections in HIV seropositive subjects. Observations from Pondicherry, India. Arora VK. Kumar SV. Indian Journal of Chest diseases and Allied sciences 41 (3): 135-44, 1999. Jul-Sep.

11. Severe Weigh Loss: The predominant Clinical presentation of tuberculosis in patients with HIV infection in India. Hira SK, Dupont HL, Lanjewar DN. Dholakia YN. National Medical Journal of India 11(6) 256-8, 1998, Nov-Dec.

12. The sero-prevalence of HIV infection among tuberculosis patients (letter). Ramachandran R. Datta.M. Shanmugam S. Bhaskar G. Subramanian R. Rawoof A. Prabhakar R. International Journal of Tuberculosis and Lung disease. 2(5); 438, 1998, May.

13. Characterisation of mycobacterial species in clinically diagnosed cases of pulmonary tuberculosis and their HIV status. Patwardhan Ns, Bansal MP. Pradhan J, Indian Journal of Pathology and Microbiology, 40(3); 365-7, 1997 Jul.

14. Impact of Human immunodeficiency virus infection on abdominal tuberculosis in Western India.

Rathi PM, Amarapurakar DN. Parikh 35, Joshi J. Koppikar GV, Amarapurkar AD, Kalro RH

Journal of Clinical Gastroenterology 24(1); 43-8, 1997 Jan.

15. Detection of HIV infection in pulmonary tuberculosis patients. Samuel NM. Alamelu C. Jaganath K. Rajan B. Journal of the Indian Medical Association 94(9); 331-3, 1996, Sep.

16. Scottish National Survey of tuberculosis notification 1993 with special reference to the prevalence of HIV seropositively. Heitch AG. Rubilar M. Curnow J Boyd G. Forbes GI. Burns S. Watt B. Thorax 51(1): 78-81, 1996 Jan.

17. Trend of HIV infection in patients with pulmonary tuberculosis in South India. Solomon S. Anuradha S. Rajasekaran S. Tubercle and Lung Disease 76(1); 17-9, 1995 Feb.

18. Tuberculosis and the HIV epidemic. Increasing annual risk of tuberculosis infection in Kenya, 1986-1996. Odhiambo Ja. Borgdortt.MW. Kiambiah FM. Kibuga DK. Kwamanga DO American Journal of Public Health 86(7); 1078-82, 1999 Jul.

19. Childhood human immunodeficiency virus and co-infections: reconciling conflicting data (Review).

    Coovadia HM Jeena P. Wilkinson D. International Journal of Tuberculosis and Lung Disease 2 (10:844, 51, 1998, Oct.).

20. Lack of direct correlation between CD 4 T lymphocytes counts and induration sizes of the tuberculin skin test in human immunodeficiency virus type 1, seropositive patients. Diagbouga S. Fumoux F. Ledru E. Sanou PT. Barro D. Machal G. Internation Journal of Tuberculosis and Lung Disease 2(4); 317-23, 1998 Apr.

21. WHO (2004) The world Health Report 2004, Report of the Director General WHO.

22. WHO (2004) Weekly Epidemiological Record 23rd Jan-2004, No.4.

23. WHO (2004) Health Development in the South East Asia region. An overview Ed. by Dr. Uton Muchtar Refei.

24. WHO (2000) Joint Tuberculosis Programme Review, India, February 2000, Regional Office for South East Asia, New Delhi.

25. WHO (2002) Global Tuberculosis Control surveillance planning financing WHO Report, 2002.

26. WHO (1999) Global Tuberculosis Control WHO Report, 1999, Geneva.

27. Government of India, 2000, Annual Report 1999-2000, Ministry of Health and Family Welfare, New Delhi.

28. WHO (2000) Joint tuberculosis programme Review, India, February 2000 Regional Office for South East Asia, New Delhi.

29. International Journal of Tuberculosis and Lung disease 2(11); 919-25, 1998 Nov.

30. HIV testing Manual laboratory diagnosis, National AIDS Control Organisation. Ministry of Health and Family Welfare, Government of India.

31. Tuberculosis related disease and HIV. Text book of pulmonary and extrapulmonary Tuberculosis S. Satyasri. Third edition, 1998, 111-113.

32. Tuberculosis in persons with Human Immuno Deficiency Virus Infection, Philip C. Hopewell. The Medical Management of AIDS. Merle A. Sande. Paul A. Volberding. Fifth edition, 1997.

33. Seroprevalance of human immunodeficiency virus HIV infection among tuberculosis patients in Tamilnadu-Ramachandran R, Datta M, Subramani R, Baskaran G, Paramasivan CN, Swaminathan S, Indian J Medi Res. 2003 Oct; 118: 147-51.

34. HIV-1 seropositivity in Pulmonary Tuberculosis (Study of 340 cases from Marathwada), Indian J Pathol Microbiol, 1993, Oct., 36(4): 383-8.

35. UNAIDS WHO 2003 AIDS epidemic update, December 2003.

36. Naco Internet site www.naco.nic.in.

37. Current Medical diagnosis and treatment Edited by Lawrence M.Tierney, Jr. Stephen J, Mcphee and Maxine A. Papadakis, 43rd Ed.2004.

PREVALENCE OF HIV IN TUBERCULOSIS PATIENTS PROFORMA

Name of the Patient	:	Ward :
IP No.	:	DOA :
Age	:	
Sex	:	
Address	:	
Occupation	:	

SYMPTOMS

Cough	Duration
	Productive / Non Productive
	Quantity
	Colour
Haemoptysis	Frequency
	Amount
Chest Pain	
Dyspnoea	
Wheeze	
Fever	Duration
	Evening rise of temperature
Loss of Appetite	Duration
Loss of Weight	Duration
Diarrhoea	Duration
Abdominal Pain	
Abdominal Distension	
Headache, Neck Pain, Vomiting	

Joint Pain, Back Pain

Family H/o. TB

Past H/o.	TB
	Jaundice
	Diabetes Mellitus
	Accidents
	Hospitalisation
	Previous Surgery
	Blood Transfusion
Personal History	Smoking
	Alcoholism
	IV Drug abuse
Marital Status	Married / Unmarried

Sexual History

- H/o. Pre / Extra Marital contract Yes / No
- Duration
- Last contract
- Single / Multiple partners
- Commercial Sex Worker / Co Worker / Relative / Neighbour
- Genital Ulcers
- White Discharge

GENERAL EXAMINATION

- Weight
- Height

Temperature

Anaemia

Cyanosis

Clubbing

Lymphadenopathy

Tinea Versicolor

Oral Candidiasis

Hairy Leukoplakia

Kyphosis

Scoliosis

Bp

Pulse

RESPIRATORY SYSTEM

Trachea

Apical impulse

Chest movement

Percussion

Auscultation

CVS

ABDOMEN Hepatomegaly

Splenomegaly

Free Fluid

CNS

Neck Stiffnes

Spinal Tenderness

Fundus

INVESTIGATIONS

Blood - TC

DC

ESR

Hb%

Mantoux test

Size

Chest X-ray

Sputum AFB

ELISA for HIV

Rapid test for HIV

AIDS REFERENCE CENTRE, INSTITUTE OF MICROBIOLOGY, MADRAS MEDICAL COLLEGE, CHENNAI.

REQUISITION FOR HIV TEST

Name	Date
Address	
Age	
Sex	
Occupation	Marital Status
Probable Diagnosis	
Hosptial	Unit

Clinical Features - Duration	Whether Tested Before
	Date of Testing
	Place of Testing
	Type of Test

ADDITIONAL INFORMATION

H/o. Blood Transfusion	:	No. of Unit
		Dateof Last Transfusion
H/O Surgery		
H/O STD		
H/O MTP		
Last Child Birth		

Whether Spouse Tested

H/O Multiple Partner Sex Premarital

Extramarital

H/O Homosexual Practice

H/O IVD

Smoking

Alcohol

GENERAL INSTRUCTION

Assume all blood and body fluids to be potentially hazardous. Observe universal safety precautions while collecting blood. Take a sterile test tube and place a proper label with name, age, sex and unit.

- Perform proper skin disinfecting at the venipuncture site withdrawn 5 ml of blood and transfer to labeled test tube.
- Allow to stand for 30 minutes for clot to form and serum to separate
- Transport to the laboratory immediately accompanied by a duly filled up request form.
- if delay is unavoidable, transfer serum into a labeled sterile screw capped vial and refrigerate overnight.
- Transport to laboratory avoiding further delay.

S.No.	Name	Age	Sex	Duration Months	H/o Weight loss	H/o extra pulmonary symptoms	Risk factors surgery, hospitalization BL,Transfusion IV drug abuse, exposure	Marital Status	STD	General Examination	Extrapulm involvement	ESR mm/hr	Total Count	Mantoux test	CXR	Diagnosis	Sputum AFB	ELISA
1.	VANITHA	27	F	6	+	-	-	M	-	-	-	20	6800	+	BUZI	B.PTB	+	_
2.	SHARMILA	16	F	2	+	Neck swelling	-	UM	-	A	LN	10	7800	+	RUZI	PTB CTBL	-	-
3.	VADIVEL	30	M	3	+	-	-	UM	-	-	-	68	10200	+	RUZFC	PTB	+	-
4.	MOHAMED	25	M	4	+	-	-	M	-	-	-	72	8200	-	RPE LUZI	PTB RPE	-	-
5.	RANJITHKUMAR	16	M	6	+	-	-	UM	-	-	-	72	9000	+	RUZI	PTB	+	-
6.	GLADWIN	36	M	6	+	Diarrhea	E(M/CSW)	M		HM	-	58	9200	-	BDI	PTB	+	+
7.	GOKUL	18	M	3	+	-	-	UM	-	-	-	82	8000	-	RUZI RPE	PTB RPE	-	-
8.	VEERAMMAL	65	F	3	+	-	-	M	-	A	-	10	7800	-	RUZI LUZI	BPTB	-	-
9.	SARAVANA	37	M	4	+	-	-	M	-	-	-	60	7200	+	RUZI	PTB	-	-
10.	VIJI	32	M	7	+	-	-	UM	-	-	-	68	6200	+	RUZI	PTB	+	-
11.	LALITHA	25	F	1	+	-	-	UM	-	A	-	20	9200	-	RUZI LUZI	BPTB	+	-
12.	NAGARAJAN	46	M	8	+	-	-	M	-	-	-	72	6400	-	RUZI	PTB	-	-
13.	SEKAR	47	M	15	+	-	-	M	-	-	-	25	4800	-	MM	MTB	-	-
14.	NOORJAKAN	55	F	1	+	-	-	M	-	-	-	30	7700	+	RPE	TBPE	-	-
15.	NARESH	47	M	2	+	-	-	M	-	-	-	28	7800	+	RUZI	PTB	-	-

S.No.	Name	Age	Sex	Duration Months	H/o Weight loss	H/o extra pulmonary symptoms	Risk factors surgery, hospitalization BL,Transfusion IV drug abuse, exposure	Marital Status	STD	General Examination	Extrapulm involvement	ESR mm/hr	Total Count	Mantoux test	CXR	Diagnosis	Sputum AFB	ELISA
16.	KAMALAM	45	F	2	+	-	-	M	-	-	-	32	6400	-	BPE	BTB PE	-	-
17.	RAJA	42	M	4	+	-	-	M	-	-	-	28	6000	+	LUZI	PTB	+	-
18.	SELVARAJ	56	M	3	+	-	-	M	-	-	-	42	8000	-	RUZI	PTB	-	-
19.	MAYAVAN	50	M	3	+	-	-	M	-	-	-	28	6000	+	RUZI	PTB	-	-
20.	RAMAN	41	M	3	+	-	-	M	-	-	-	86	8900	+	RUZ FC	PTB	-	-
21.	DHANRAJ	27	M	3	+	-	-	UM	-	-	-	68	6800	-	RUZI	PTB	-	-
22.	KAMALA	65	F	3	+	-	-	M	-	-	-	60	4800	+	RUZI LUZI	BPTB	-	-
23.	PATCHIAPPAN	52	M	1	-	-	-	M	-	-	-	62	8000	+	RUZI RPE	PTB RPE	+	-
24.	SIVAKUMAR	26	M	3	+	-	-	UM	-	-	-	38	6500	-	RUZI	PTB	-	-
25.	CHINNASAMY	51	M	2	-	-	-	M	-	-	-	62	8000	+	RPE	RPE	-	-
26.	THULUKANNAM	60	F	2	-	-	-	M	-	-	-	25	5000	+	RUZI LUZI	BPTB	+	-
27.	MANIKAM	37	M	6	+	-	-	M	-	-	-	58	9600	-	RUZI	PTB	+	-
28.	SADHIYA	13	F	3	+	-	-	UM	-	-	-	45	8000	-	RUZI	PTB	-	-
29.	VELAN	38	M	3	+	-	-	M	-	-	-	60	6300	-	RUZI	PTB	-	-
30.	MOHAN	44	M	4	+	-	-	M	-	-	-	86	4600	-	LPE	LPE	-	-

S.No.	Name	Age	Sex	Duration Months	H/o Weight loss	H/o extra pulmonary symptoms	Risk factors surgery, hospitalization BL,Transfusion IV drug abuse, exposure	Marital Status	STD	General Examination	Extrapulm involvement	ESR mm/hr	Total Count	Mantoux test	CXR	Diagnosis	Sputum AFB	ELISA
31.	RAJESWARI	25	F	4	+	-	-	M	-	-	-	60	8000	+	RUZI LUZI	B.PTB	-	_
32.	MATHIVANNAN	24	M	4	+	SEIZURE	-	UM	-	-	CNS	62	8100	-	RPE	TBM RPE	-	-
33.	VALLIAMMA	35	F	1	-	-	-	M	-	-	-	40	4600	+	RUZI MZI	PTB	-	-
34.	RAJENDRAN	40	M	6	+	-		M		-	-	60	7000	+	RUZI MZI	PTB	+	-
35.	ELUMALAI	28	M	10	+	-	E(M.CSW	M	-	A,OC,HL	-	40	7600	-	BDI	PTB	-	+
36.	CHINNAMMAL	40	F	4	+	-	-	M	-	-	-	60	5400	-	RUZI LMZI	BPTB	-	-
37.	KAMSALA	27	F	3	+	-	-	M	-	-	-	45	6000	+	RUZI	PTB	+	-
38.	RAMESH	21	M	7	+	-	-	UM	-	-	-	56	5600	-	RUZI	PTB	+	-
39.	ARUMUGAM	42	M	2	-	-	-	M	-	-	-	28	6800	+	RUZI MZI	PTB	-	-
40.	SAROJA	40	F	2	-	-	-	M	-	-	-	50	6500	+	RUZI MZI	PTB	+	-
41.	SANDOSH	28	M	4	+	-	-	UM	-	-	-	60	7200	-	RUZI	PTB	-	-
42.	PADMA	28	F	10	+	ABD DIS	-	M	-	-	ABD	60	6800	+	RUZI	PTB TBAB	+	-
43.	MURUGAN	39	M	6	+	-		M	-	-	-	56	6600	-	RUZI	PTB	-	-
44,	VASANDHARAJ	42	M	7	+	-	-	M	-	-	-	40	8000	+	LUZI	PTB	+	-
45.	THIRUPURA SUNDARI	50	F	10	+	-	-	M	-	-	-	45	7200	+	RUZI LUZI	BPTB	-	-

S.No.	Name	Age	Sex	Duration Months	H/o Weight loss	H/o extra pulmonary symptoms	Risk factors surgery, hospitalization BL,Transfusion IV drug abuse, exposure	Marital Status	STD	General Examination	Extrapulm involvement	ESR mm/hr	Total Count	Mantoux test	CXR	Diagnosis	Sputum AFB	ELISA
46.	ELLAMMAL	60	F	1	-	-	-	M	-	-	-	30	6800	+	RUZI LUZI	B.PTB	-	_
47.	MURALI	32	M	2	-	-	-	UM	-	-	-	54	8100	+	RPE RUZI	PTB RPE	+	-
48.	DEENADYALAN	24	M	6	+	-	-	M	-	-	-	58	7100	-	RUZI	PTB	-	-
49.	SURIYAN	42	M	2	+	PARA PLEGIA	-	M	-	-	SPINE	65	8000	-	N	TB SPINE	-	-
50.	GOPAL	44	M	2	+	-	-	M	-	-	-	82	8100	-	RPE	RPE	-	-
51.	MANJU	38	F	6	+	-	-	M	-	-	-	30	6800	+	RUZI MZI	PTB	-	-
52.	PERUMAL	30	M	5	+	-	E(MCSW)	M		HM, A, TV	-	70	10000	-	BPI	PTB	-	+
53.	SAKTHI	40	M	3	+	-	-	M	-	-	-	48	7200	-	RUZI	PTB	-	-
54.	DIVIYA	13	F	150	-	SEIZURE	-	UM	-	-	-	60	9000	-	N	TBM TUBER	-	-
55.	ANNAMALAI	38	M	2	-	-	-	M	-	-	-	58	8200	+	RUZI	PTB	+	-
56.	GANGA	32	F	6	+	-	-	M	-	OC, HL	-	15	6200	-	BDI	PTB	-	+
57.	LALITHA	45	F	3	+	-	-	M	-	-	-	40	5600	+	RUZI MZI	PTB	+	-
58.	PALANIVEL	37	M	1	-	-	-	M	-	-	-	60	8100	+	RUZI	PTB	-	-
59.	MURUGAIYA	31	M	2	+	-	-	M	-	-	-	82	6800	-	RPE	RPE	-	-
60.	GEETHA	14	F	4	+	-	-	UM	-	-	-	62	8000	-	RPE RUZI	-	-	-

S.No.	Name	Age	Sex	Duration Months	H/o Weight loss	H/o extra pulmonary symptoms	Risk factors surgery, hospitalization BL,Transfusion IV drug abuse, exposure	Marital Status	STD	General Examination	Extrapulm involvement	ESR mm/hr	Total Count	Mantoux test	CXR	Diagnosis	Sputum AFB	ELISA
61.	RUCKMANI	60	F	4	+	ABD DIS	-	M	-	A	-	63	8200	+	RUZI MZI	PTB TB.AB4	-	-
62.	PARVATHI	64	F	2	+	-	-	M	-	-	-	64	8000	-	RPE RUZI	PTB RPE	-	-
63.	NAGAMMAL	65	F	4	+	-	BT	M	-	A	-	84	7200	-	RUZI LUZI	BPTB	-	-
64.	MURUGESAN	29	M	1	+	Diarrhea	E(M/CSW)	M		A, TV, DPT, Icty	-	60	8800	-	RUZFC BMZI	BPTB	-	-
65.	GANESH	25	M	4	+	-	-	UM	-	-	-	56	8000	+	RUZI	PTB	-	-
66.	RAJESWARI	37	F	ISD	-	Head ache	-	M	-	-	CNS	72	68000	-	RUZI MZI	PTB TBM	-	-
67.	SENTHIL	27	M	6	+	-	-	UM	-	-	-	56	6500	-	RUZI	PTB	+	-
68.	BALARAMAN	53	M	7	+	-	-	M	-	-	-	65	7000	-	RUZI	PTB	+	-
69.	DEVI	55	F	6	+	-	-	M	-	-	-	22	4800	-	RUZI	PTB	-	-
70.	RAVI	40	M	2	+	-	-	M	-	-	-	72	6800	+	RUZI MZI	PTB	-	-
71.	BABU	40	M	1	-	-	-	M	-	-	-	25	8000	-	MM	MTB	-	-
72.	IBRAHIM	30	M	1	+	-	-	UM	-	-	-	68	8200	-	R Pneumo	R Pneumo	-	-
73.	VENTESH	54	M	3	+	-	-	M	-	-	-	68	7100	-	RUZI	PTB	-	-
74.	KARTHIKEYAN	26	M	4	+	-	-	UM	-	-	-	72	6100	-	RUZI	PTB	-	-
75.	JANETRANI	13	F	200	-	Cervical adeno pathy	-	UM	-	-	LN	72	8000	+	RUZI MZI	PTB CTBL	+	-

S.No.	Name	Age	Sex	Duration Months	H/o Weight loss	H/o extra pulmonary symptoms	Risk factors surgery, hospitalization BL,Transfusion IV drug abuse, exposure	Marital Status	STD	General Examination	Extrapulm involvement	ESR mm/hr	Total Count	Mantoux test	CXR	Diagnosis	Sputum AFB	ELISA
76.	KANNIGA	35	F	20D	-	SEIZURES	-	M	-	A	CNS	40	9600	+	N	TBM	-	_
77.	VEERASAMY	48	M	3	+	-	-	M	-	-	-	40	6000	-	MM	MTB	-	-
78.	MUTHURAJ	32	M	10	+	DIARRHOEN	EMCSW	UM	-	A Scabics	-	44	8600	-	RPLE	RPE	-	+
79.	DOSS	57	M	4	+	-	-	M	-	-	-	30	6300	-	RUZI	PTB	+	-
80.	CHITRA	25	F	15D	-	Seizures	-	UM	-	-	CNS	60	13200	-	RUZI	PT TBM	-	-
81.	MARY	40	F	3	+	-	-	M	-	-	-	15	4600	-	RUZI LUZI	BPTB	+	-
82.	MUNIANDI	32	M	3	+	-	-	M	-	-	-	42	7400	+	RUZI	PTB	-	-
83.	SINGARAJ	29	M	4	+	-	-	M	-	-	-	38	6600	-	RUZI	PTB	-	-
84.	MASILAMANI	52	M	4	+	-	-	M	-	-	-	42	4800	+	LUZI	PTB	+	-
85.	SALAMMAL	56	F	1	+	-	-	M	-	A	-	25	7500	-	MM	MTB	-	-
86.	SIVASHANKAR	30	M	5	+	-	-	UM	-	-	-	62	6300	-	RUZ FC	PTB	-	-
87.	SHANKAR	40	M	2	+	-	-	M	-	-	-	68	9400	+	RUZI	PTB	+	-
88.	SHANDHA	45	F	6	+	-	-	M	-	-	-	12	4600	+	RUZI MZI	PTB	+	-
89.	RAJA	44	M	3	+	-	-	M	-	-	-	62	9000	-	RUZ FC	PTB	-	-
90.	ALAMELU	26	F	2	+	ABDOMI NAL DISTENTION	-	M	-	A	ABDOMEN	13	6500	+	LUZI	PTB TB ABD	-	-

S.No.	Name	Age	Sex	Duration Months	H/o Weight loss	H/o extra pulmonary symptoms	Risk factors surgery, hospitalization BL,Transfusion IV drug abuse, exposure	Marital Status	STD	General Examination	Extrapulm involvement	ESR mm/hr	Total Count	Mantoux test	CXR	Diagnosis	Sputum AFB	ELISA
91.	SOUNDARAMMA	40	F	3	+	-	-	M	-	-	-	62	5000	-	RE LUZI	PTB RPE	-	_
92.	SURESH	27	M	3	+	-	-	UM	-	-	-	68	9700	-	RUZI MZI	PTB	-	-
93.	DHANABAL	60	M	4	+	-	-	M	-	-	-	58	4600	+	RPE	RPE	-	-
94.	RAGUNATHAN	55	M	3	+	-	-	M	-	-	-	60	8100	+	RUZI LUZI	BPTB	+	-
95.	SAROJA	45	F	6	+	-	-	M	-	-	-	26	7100	+	RUZI MZI	PTB	-	-
96.	JOHN	52	M	3	+	-	-	M	-	-	-	42	7800	-	RPE	TB RPE	-	-
97.	KAMARUDEN	47	M	6	+	-	-	M	-	-	-	62	6000	+	RUZI MZI	PTB	-	-
98.	SHANKAR	36	M	3	+	-	-	M	-	-	-	42	8000	-	RPE	TB RPE	-	-
99.	NALINI	24	F	2	+	-	-	UM	-	-	-	56	8100	-	RUZI MZI	PTB	-	-
100.	KAMATCHI	40	F	4	+	-	-	M	-	-	-	60	8000	-	RUZI	PTB	+	-

DESCRIPTION OF POSITIVE CASES

Sl.No.	I.P.No.	Age	Sex	Duration of symptoms months	Extra pulm Symptoms	Risk factors	Marital Status	Clinical marker	ESR (mm/hr)	Total Count	CXR	MX	AFB
1.	768958	36	M	6	Diarrhea	E(M/CSW)	M	HM	58	9200	BDI	-Ve	+Ve
2.	762935	28	M	10	-	E(M/CSW)	M	A, OC, HL	40	7600	BDI	-Ve	-Ve
3.	786920	30	M	5	-	E(M/CSW)	M	HM, A, TV	70	10,000	BDI	-Ve	-Ve
4.	778643	29	M	1	Diarrhea	E(M/CSW)	M	TV,ICY,A,DPT	60	8800	RUZFC/ BMZI	-Ve	-Ve
5.	743202	32	F	6	-	E (M)	M	OC-HL	150	10000	BDI	-Ve	-Ve
6.	776714	32	M	10	Diarrhea	E (M/CSW) GU	M	A,TV, scabies	44	8800	RT, PLEF	-Ve	-Ve

I.P.No. - In Patient Number
E - Exposure
S - Single
M - Multiple
CSW - Commercial Sex Worker
GU - Genital Ulcer

ICY - Iethyosis
UM - Unmarried
M - Married
HM - Hepatomegaly
A - Anaemia
TV - Tinea Versicolar

DPT - Dirty Pigmented tongue
OC - Oral Candidiasis
HL - Hairy Leukoplakia
CXR - Chest X-ray
UZ - Upper Zone
MZ - Mid Zone

BDI - Bilateral Diffuse Infiltrates
PLEF - Pleural Effusion

ABBREVIATIONS USED IN MASTER CHART

STD	-	Sexually Transmitted Diseases
ESR	-	Erythrocyte Sedimentation rate
CXR	-	Chest X - ray
AFB	-	Acid Fast Bacilli
D	-	Days
CNS	-	Central Nervous System
J	-	Jaundice
M	-	Multiple
CSW	-	Commercial Sex Worker
TBM	-	Tuberculous Meningitis

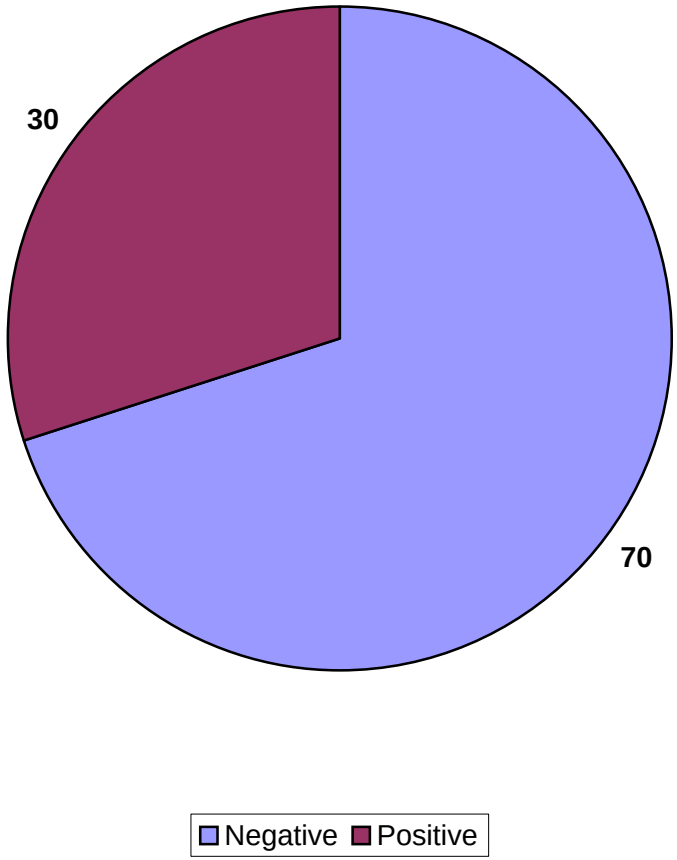
Martial Status

M	-	Married
UM	-	Unmarried
A	-	Anaemia
TBAB	-	Tuberculour Abdomen
L	-	Lymphadenopathy
TV	-	Tinea Versicolar
CTBL	-	Cervical TB Lymphadenitis
HM		Hepatomegaly

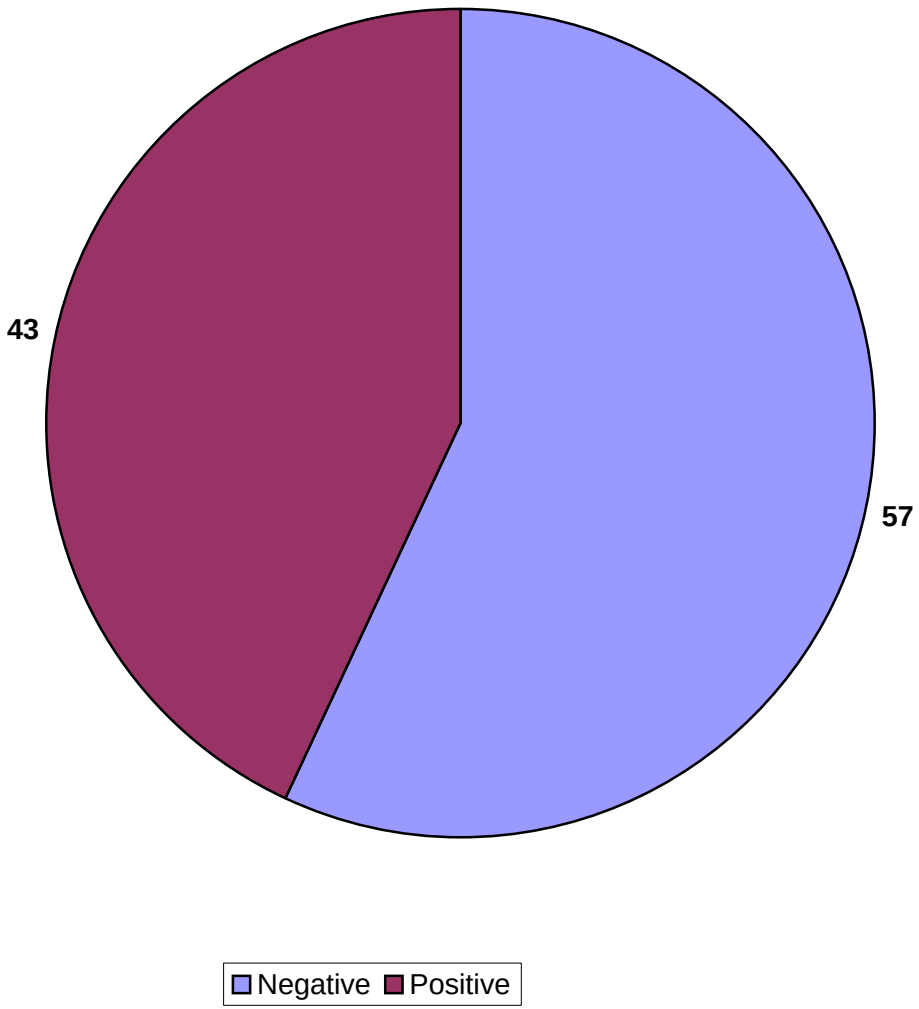
UZ	-	Upper Zone
MZ	-	Mid Zone
LZ	-	Lower Zone
I	-	Infiltrate
D	-	Diffuse
PE	-	Pleural Effusion
C	-	Cavity
N	-	Normal
Pneumo-		Pneumothorax

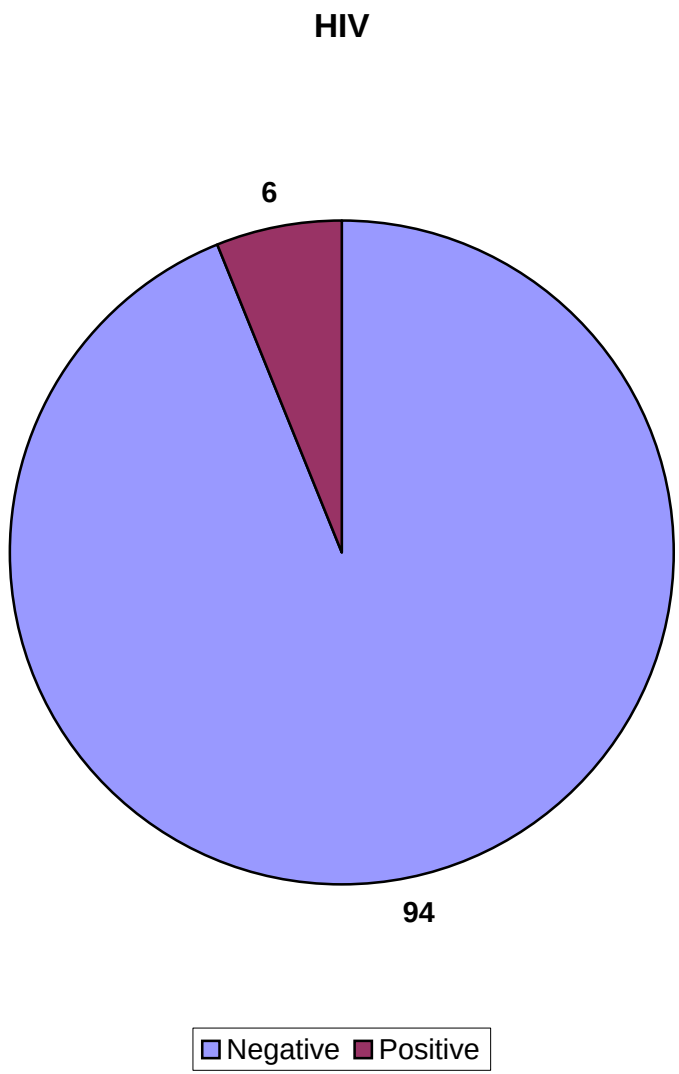
MTB	-	Miliary Tuberculosis
DPT	-	Dirty Pigmented Tongue
HL	-	Hairy LEUkoplakia
B	-	Bilateral
R	-	Right
L	-	Left
MM	-	Miliary Mottling
OC	-	Oral Candidiasis

SPUTUM AFB



MANTOUX TEST



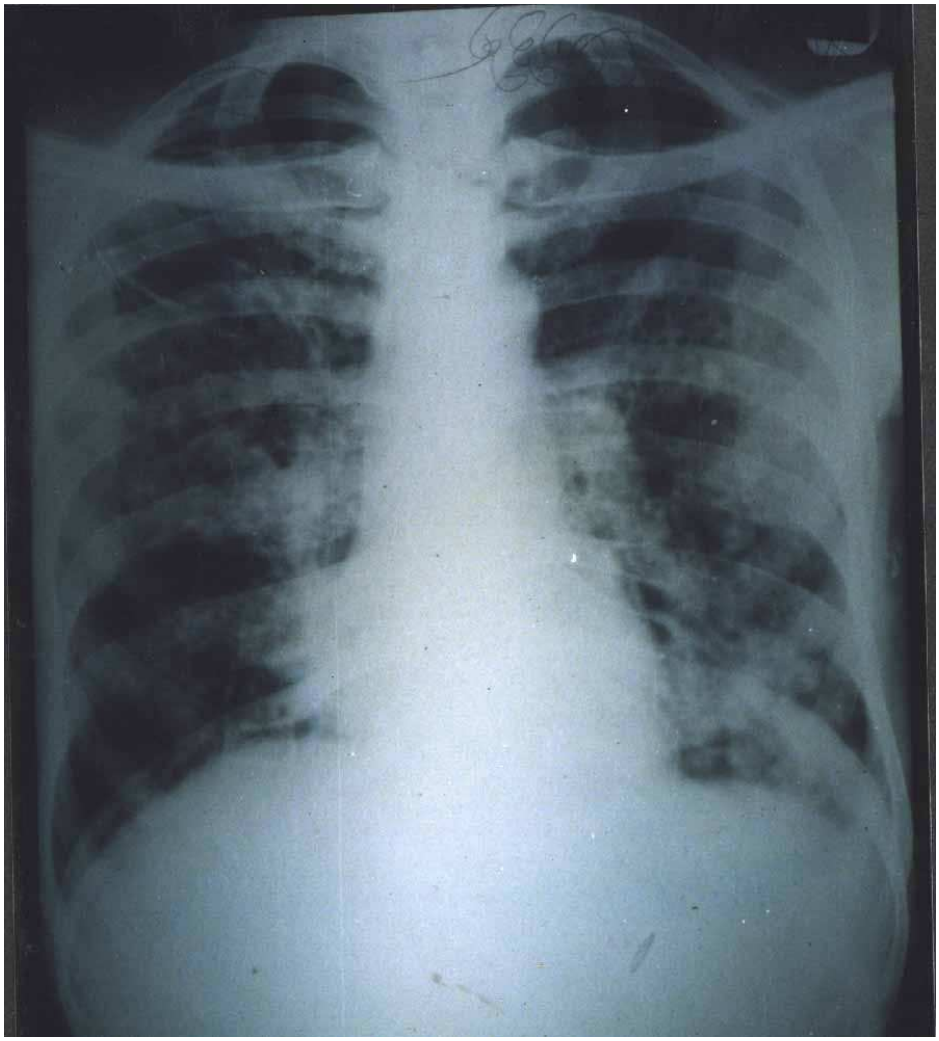

HIV
6
94
Negative
Positive

Negative	Positive
70	30

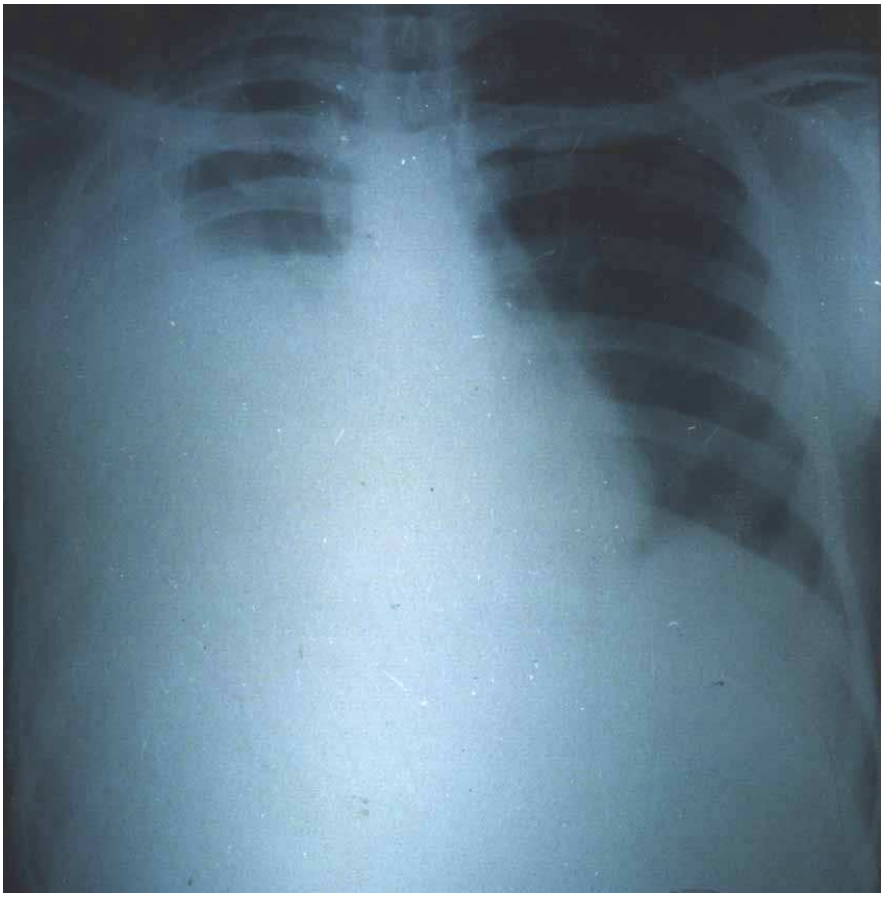
Negative	Positive
57	43

Negative	Positive
94	6

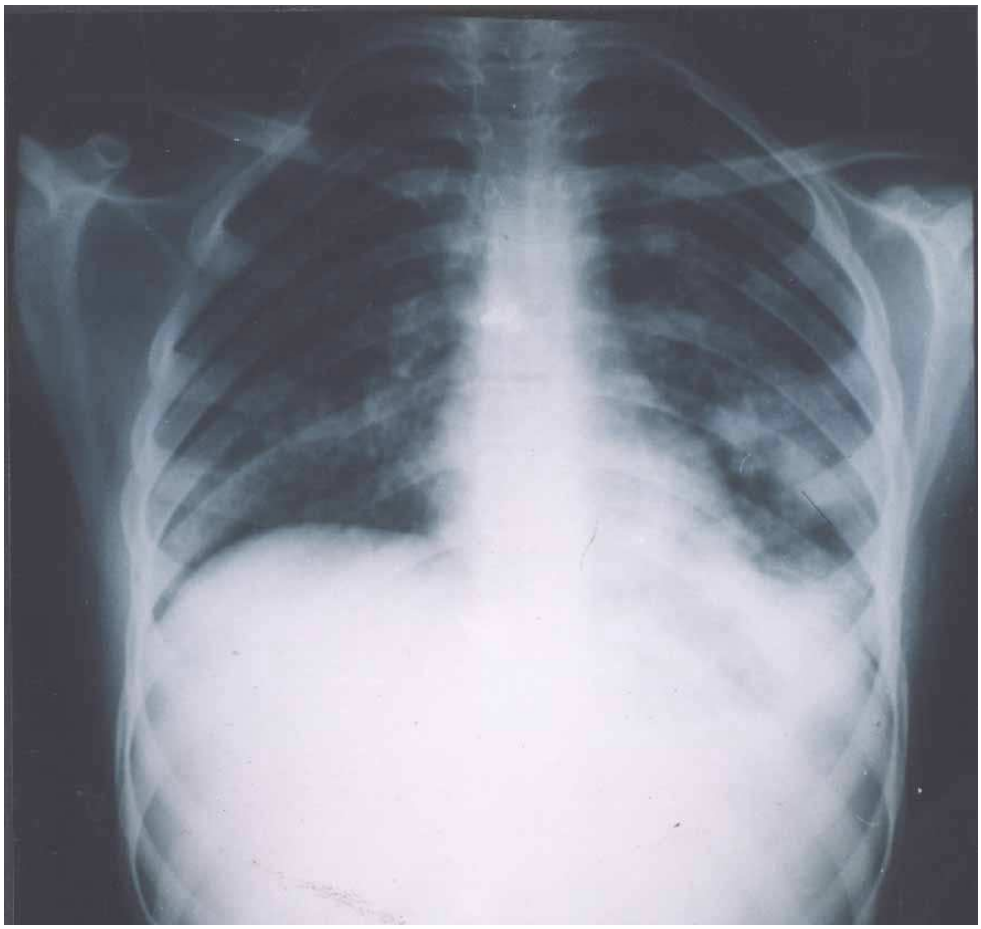
HIV POSITIVE PATIENT -
EXTENSIVE PULMONARY TUBERCULOSIS



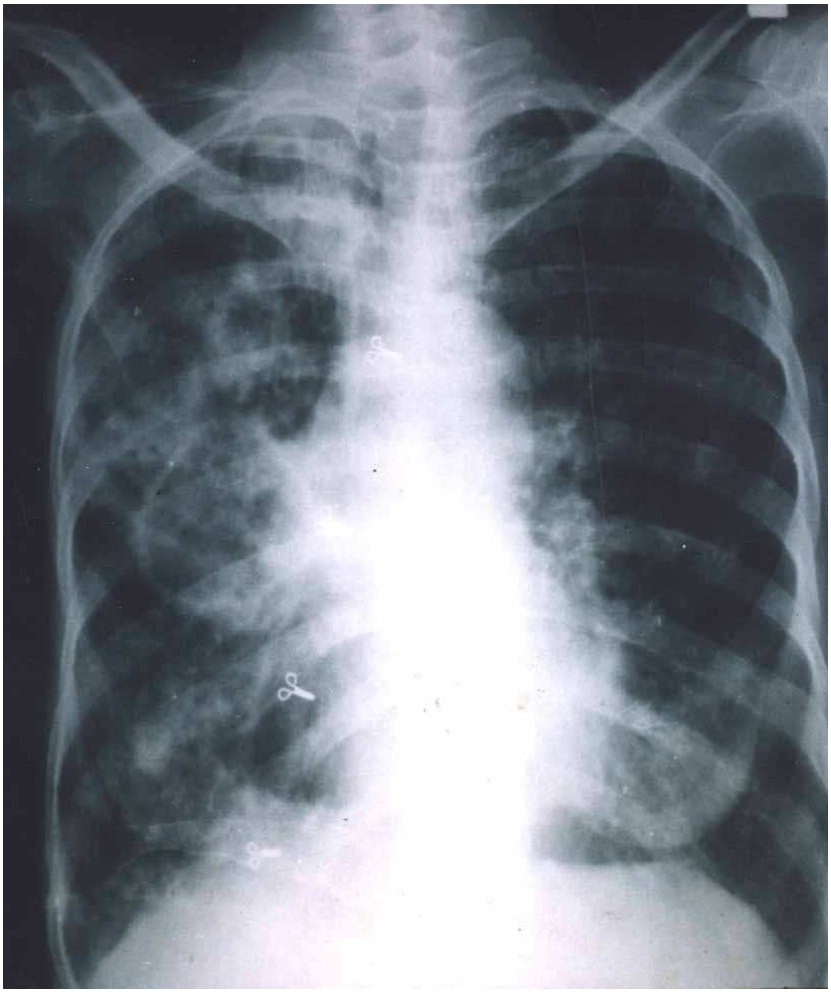
HIV POSITIVE PATIENT -
RIGHT PLEURAL EFFUSION



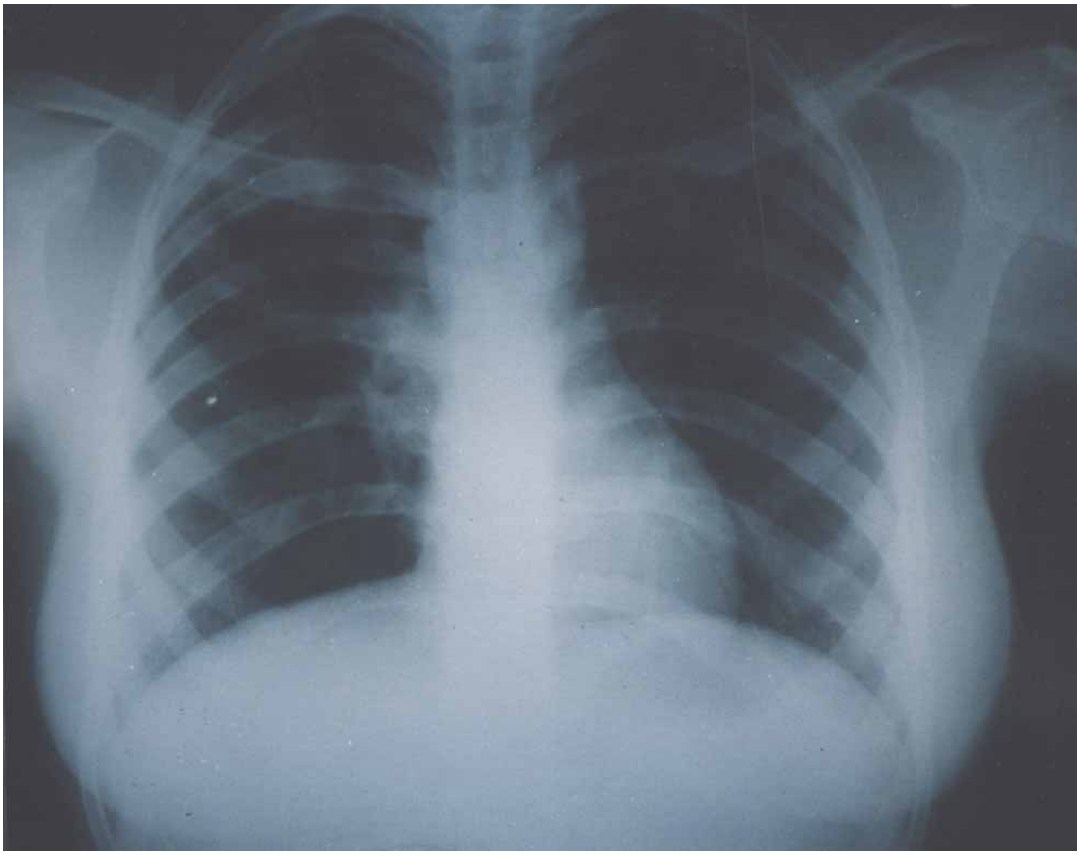
HIV POSITIVE PATIENT -
EXTENSIVE PULMONARY TUBERCULOSIS



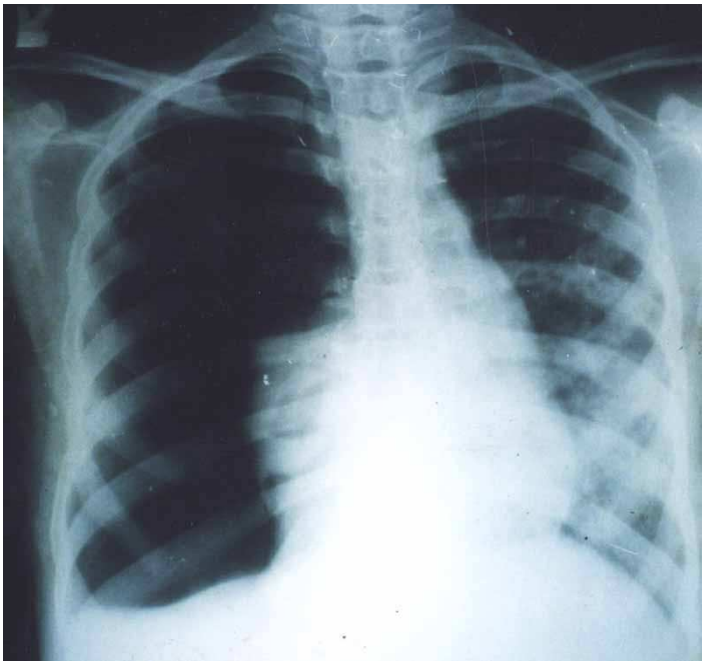
HIV POSITIVE PATIENT -
RIGHT MULTIPLE CAVITIES



HIV NEGATIVE PATIENT-
RIGHT UPPER ZONE CAVITY



**HIV NEGATIVE PATIENT -
RIGHT PNEUMOTHORAX, BEFORE
INTERCOSTAL DRAINAGE**



**HIV NEGATIVE PATIENT -
RIGHT PNEUMOTHORAX,
AFTER INTERCOSTAL DRAINGAGE**